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SURGERY

Medical History, Humanism, and the Student of Medicine
Henry R. Viets

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Richard J. Field Sr. and Richard J. Field Jr.

Further Studies on the Question of an Inhibitory Hormone from the Gastric Antrum
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Lowell Brown and Harry Essig

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Surgical Treatment of Thrombotic Occlusion of Aorta and Iliac Arteries
John M. Erskine, Frank L. Gerbode, S. W. French III, and R. Maurice Hood

Treatment of Squamous-Cell Carcinoma of the Floor of the Mouth
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*Brown, S. S.; Libo, H. W., and Nussbaum, A. H.: Norethandrolone in the Successful Management of Anorexia and "Weight Lag" in Children, Scientific Exhibit presented at the Annual Meeting of the American Academy of Pediatrics, Chicago, Oct. 20-23, 1958.

TABLE OF CONTENTS

VOLUME 79 JULY 1959 NUMBER 1

ORIGINAL ARTICLES

PAGE

Medical History, Humanism, and the Student of Medicine
Henry R. Viets, M.D., Boston 1

Intestinal Obstruction Produced by Fecalith Arising in Meckel's Diverticulum
Richard J. Field Sr., M.D., and Richard J. Field Jr., M.D., Centreville, Miss. 8

Further Studies on the Question of an Inhibitory Hormone from the Gastric Antrum
Lester R. Dragstedt, M.D., Ph.D.; Soichi Kohatzu, M.D.; James Gwaltney, M.D.; Kazuo Nagano, M.D., and Herbert B. Greenlee, M.D., Chicago, with the technical assistance of Donald B. Thurs[illegible] and Richard A. Weinberg. 10

Small-Intestinal Gallstone Ileus and Use of Barium Sulfate per Os in Its Diagnosis
Victor Drucker, M.D., Binghamton, N. Y. 22

Closed-Chest Resuscitation
Robert M. Hosler, M.D., and Kenneth Wolfe, Cleveland 31

A Suction Apparatus for Use During Open Cardiotomy
Elton Watkins Jr., M.D., Boston, and Comdr. Alexander C. Hering (MC), U. S. N., with the technical assistance of Mary Ann Dalton, R.N., and Ralph W. Sommers 35

Idiopathic Hyperlipemia
Major Raimonds J. Gailitis (MC), U. S. Army, and Werner Schreiber, Dr. Med., Nürnberg, Germany 40

Tracheotomy in Burns
John A. Moncrief, M.D., Atlanta 45

Subdural Hematoma Following Spinal Anesthesia
Keasley Welch, M.D., Denver 49

Some Problems Encountered in Treatment of Peripheral Arterial Disease
Thomas G. Baffes, M.D.; Clarence Norberg, M.D., and Spyros Katopodis, M.D., Chicago 52

Use of Pneumoperitoneum in Differential Diagnosis of Paracardiac Masses in Children
David H. Baker, M.D.; Bernard Maisel, M.D., and Henry P. Goldberg, M.D., New York 63

An Amazing Accident: Traumatic Transection of Bronchus and Aorta
LaMoyne C. Bleich, M.D., Ruston, La. 68

Fatal Rupture of an Ivalon (Polyvinyl Formalinized) Sponge Aortic Graft into Duodenum
Lowell Brown, M.D., and Harry Essig, M.D., Freeport, L. I., N. Y. 72

Intranasal Encephalomeningoceles Associated with Cranium Bifidum
Henry W. Dodge Jr., M.D.; J. Grafton Love, M.D., and James W. Kernohan, M.D., Rochester, Minn. 75

Surgical Treatment of Thrombotic Occlusion of Aorta and Iliac Arteries
John M. Erskine, M.D.; Frank L. Gerbode, M.D.; S. W. French III, M.D., and R. Maurice Hood, M.D., San Francisco 85

Treatment of Squamous-Cell Carcinoma of the Floor of the Mouth
John B. Erich, M.D., and Lyle V. Kragh, M.D., Rochester, Minn. 94

Results of Treatment of Squamous-Cell Carcinoma Arising in Mandibular Gingiva
John B. Erich, M.D., and Lyle V. Kragh, M.D., Rochester, Minn. 100

Severe Gastrointestinal Hemorrhage Resulting from Recklinghausen's Disease
Turley Farrar, M.D., and Fenwick W. Chappell, M.D., Memphis, Tenn. 106

Sliding Inguinal Hernia in Female Infants
Stephen L. Gans, M.D., Beverly Hills, Calif. 109

Primary Carcinoma of the Liver
Capt. David P. Minichan Jr. (MC), U. S. A. F.; Robert L. A. Keeley, M.D.; Hugh H. Trout Jr., M.D., and Daniel Leavitt, M.D., Roanoke, Va. 114

The Thyroid Nodule
G. C. Freeman, M.D., Honolulu 129

(Continued on next page)

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VOLUME 79 JULY 1959 NUMBER 1

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TABLE OF CONTENTS (Continued)

The Splenic Artery
Robert I. Lowenberg, M.D., New Haven, Conn., with the technical assistance of Anthony Piccirillo and James Nora 135

EDITORIAL

Postoperative Wound Infections
J. E. Strode, M.D., Honolulu 141

CASE REPORTS

Triplication of Gallbladder
Welland A. Hause, M.D., Decatur, Ill. 144

Sigmoid Colon Pull-Through Procedure for Rectovaginal Fistula
W. W. Glas, M.D., Eloise, Mich. 146

Benign Cyst of the Adrenal Gland
Harry V. Armitage, M.D., Chester, Pa. 148

Metastatic Chordoma
Austin J. McSweeney, M.D., and P. Richard Sholl, M.D., Janesville, Wis. 152

Kartagener's Syndrome
Lieut. Col. Billie G. Streete (MC) and Major Fred E. Stull Jr. (MC), U. S. A. F. .. 156

Choledochal Cyst Treated by Sphincterotomy
Carl E. Andrews, M.D., and Willard F. Ande, M.D., West Palm Beach, Fla. 158

Papillary Adenoma of the Ampulla of Vater
James A. Rock, M.D., and Edwin R. Fisher, M.D., Pittsburgh 163

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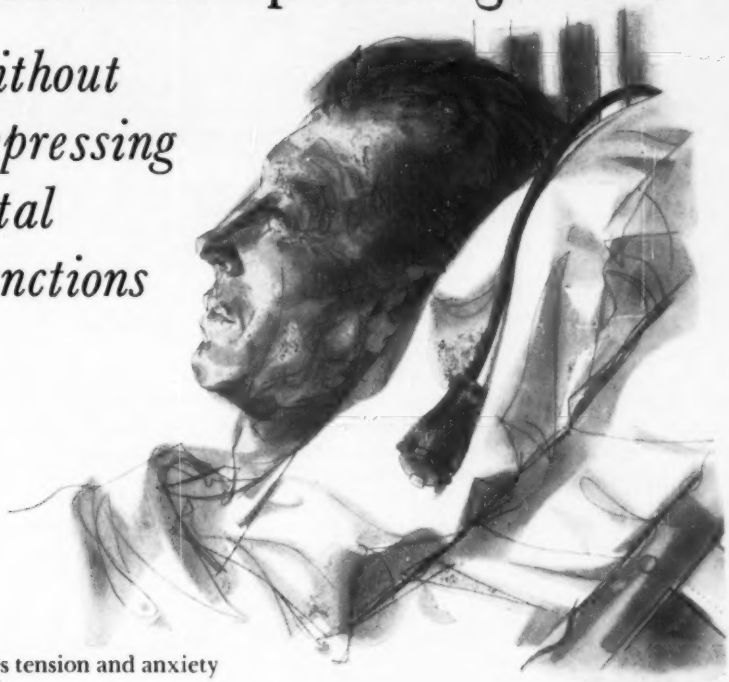

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5. Rushia, E. L.: Preliminary report on the use of meprobamate for pre-anesthetic sedation. J.M.A. Georgia 46:93, March 1957.
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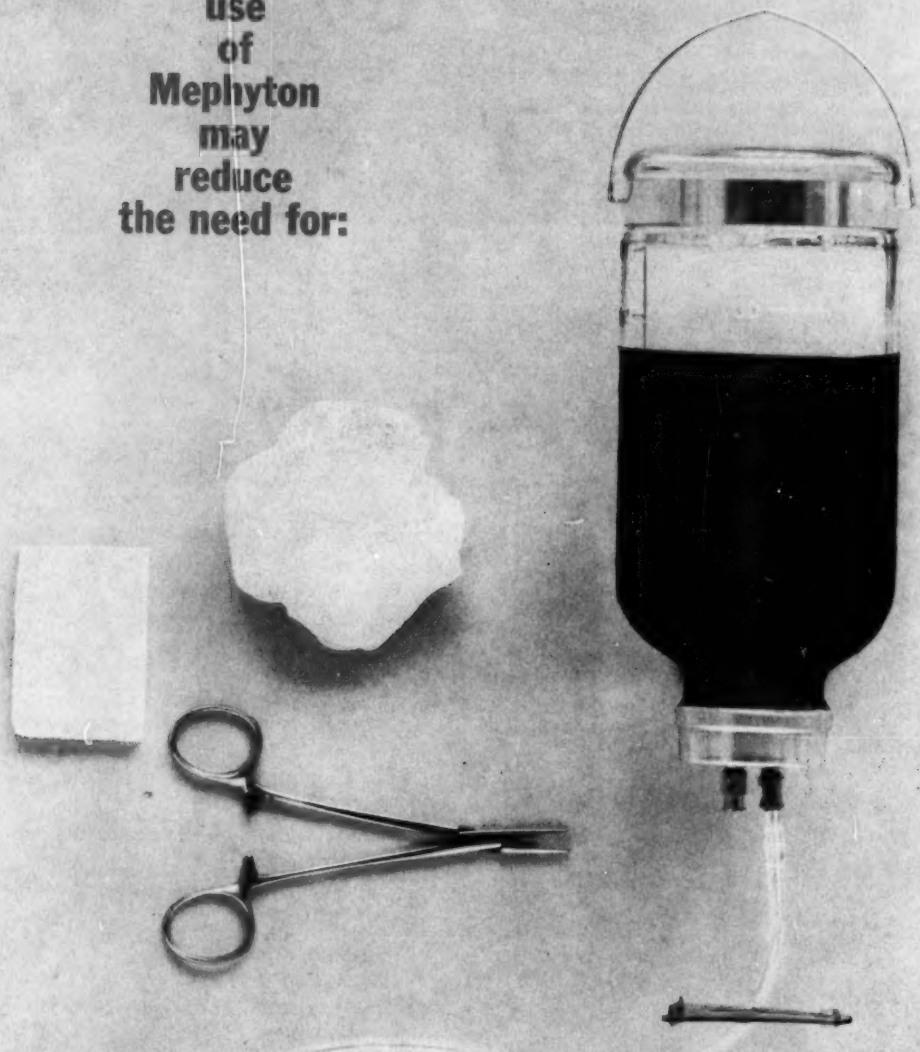

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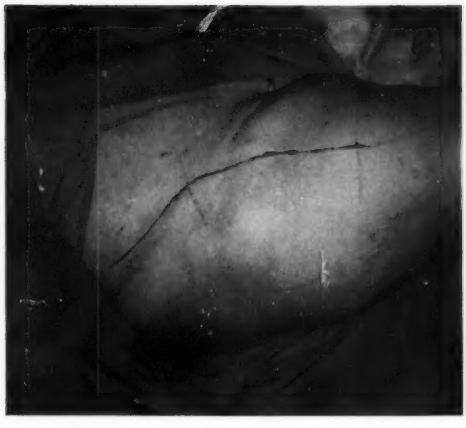

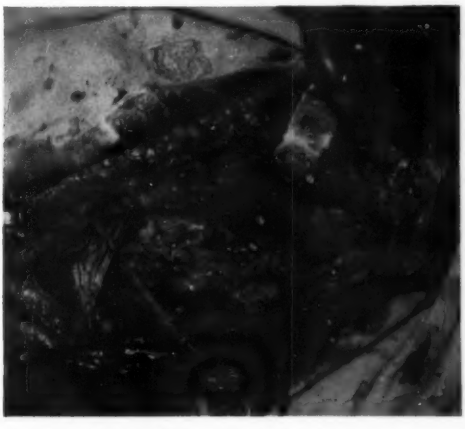

1. *(Illustration at left)* Anterior iliofemoral incision. Gloved hand is on medial side. **2.** *(Right)* Joint is now exposed. See how clearly color brings out the details: anterior capsule at bottom of wound; iliac crest at top; lateral femoral cutaneous nerve crossing below retractors.

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3. Capsule opened shows synovitis and irregularity of the osteocartilaginous junction of the head and neck.

4. Head of femur has now been dislocated. Note erosion of cartilage in the acetabulum.

5. Acetabular reamer positioned for cleaning cartilage from acetabulum. *Here color differentiates textures.*

6. Head of femur is being cleaned of all sclerotic and cystic substandard bone and degenerated cartilage.

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7. Shows cup on head of femur.

8. Hip, reduced, back in position prior to closing incision.

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Medical History, Humanism, and the Student of Medicine

HENRY R. VIETS, M.D., Boston

Foreword

Even though this journal is concerned primarily with the publication of papers on the practice of surgery and surgical research, the editors from time to time have published noteworthy papers dealing with the philosophy of the practice of medicine in its broadest aspects. For example, papers have been presented which dealt with problems in the related medical sciences. Other presentations have treated of surgical teaching, investigation and research in the Armed Forces Medical Services and the Veterans Administration. This policy of extensive inclusion has enabled us to disseminate widely among the civilian readers of this journal valuable information about the activities of our colleagues in these and other branches of the government's medical services and thus to coordinate many useful data with data furnished by their civilian counterparts.

Recently the noted neurologist, Dr. Henry R. Viets, curator of the Boston Medical Library, presented a paper at Dartmouth College on "Medical History, Humanism, and the Student of Medicine." This paper contained such important advice to those embarking on a career in medicine, as well as to those actively engaged in medicine, that its philosophy seemed worthy of wide dissemination—and where could there be a better place to accomplish such an objective than in this journal?

It gives us great pleasure to publish the thoughts of such a noted physician and scholar as Dr. Viets in this issue of the A. M. A. ARCHIVES OF SURGERY.

WALTMAN WALTERS, M.D.

The honor of giving the Mayo Memorial Lecture at Dartmouth College is not one that can be taken lightly, for we must think in terms of two of the greatest physicians in American medicine. From a small clinic developed by their father shortly before 1900, Doctor Will and Doctor Charles, as they were always known to their contemporaries, established the Mayo Clinic in Rochester, Minn., and later the Mayo Foundation for Medical Education. Expert craftsmen in surgery, Doctor Will, the elder, cultivated the field of abdominal surgery; his younger brother, Doctor Charles, paid particular attention to the treatment of goiter, a disease prevalent in the Midwest. These country doctors established a clinical and educational center, soon to become known throughout the United States and ultimately around the world. The brothers, however, were not satisfied with just serving their community as competent surgeons; they also thought in terms of raising the standards of surgery, and indeed of all medicine, to a height hardly equalled in modern times. Although devoting their

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energies primarily to improving the care of patients, they combined this endeavor with a plan to educate doctors who ultimately could go to the far corners of the earth carrying the stamp of the Mayo Clinic. Indeed, Dartmouth College and its Medical School has not been free from this endorsement. Thanks to the vision of the brothers Mayo, the world has become healthier and a better place to live and the label they gave to their achievement is still an ever-expanding influence on the progress of medicine.

Passing from one phase of life into another is far from a new experience for man, and you, as students of medicine, now bridging the gulf between an academic sojourn and a somewhat different journey into an environment of science and the art of the practice of medicine, need have no fear of the stability of the link or your ability to pass safely over it. The gulf is not so wide as many suppose, for the same principles of social conduct and the acquisition of knowledge, however unfamiliar the new language and the surroundings of a medical school, pose no iron curtain of complete exclusion of the one as you enter the other. Indeed, what you have already learned in college will serve as a substantial base on which to build your medical career. Without a secure foundation in the essentials of living, the easiness of social interplay in association with your fellows, the selective curiosity that goes with an open mind, the ability of learning how to avoid the haphazard and fortuitous situations occurring with such irritating regularity in our daily tasks, the development of Darwin's "serviceable association habits"—without these, I say, you run the risk of becoming a creature of fact and fantasy, or worse still, a spineless drifter without self direction. Too often in a nose-to-the-grindstone academic course the importance of this basic equipment is underemphasized and the door is not left widely open for the continued interpenetration and coherence of the fundamental unifying elements of history and culture.

When I was in college, for instance, I became interested in Shakespeare's "Hamlet," and spent a year studying this play.[1] The instruction was given, as not uncommon forty years ago, in a Who-What-When course, so that at the end I was more than qualified to take part in a quiz program not dissimilar to those now seen on television. I knew what was said, when, and who said it, but except for a little glimpse of the beauty of the language no clue was given toward an understanding of the dramatic significance of the dialogue, or as to how each word fitted into the working of the plot as Shakespeare pulled the strings of his puppet show to create the greatest illusion in literature. No one discussed the significance of the words of Polonius to the King and Queen, "I'll loose my daughter to him," or what destination Hamlet had in mind for Ophelia when he urged her, "Get thee to a nunnery." Both quotations are keys to an understanding of Hamlet's brutal conduct towards a seemingly innocent girl, yet no hint was suggested to help me unlock this puzzle box. The seed, however, was planted in 1909 and my curiosity aroused, although I had to wait many years before finding satisfactory answers to those and many other questions.

The disclosure came in London in 1934. Concerning "Hamlet," it was the publication of a new edition by the Cambridge University Press, edited by John Dover Wilson,[2] that gave to me for the first time adequate answers to the many questions raised in my mind when I was in college twenty-five years before.[3] Dover Wilson, moreover, expanded his ideas about the play the next year in another book, "What Happens in Hamlet."[4] These two books have been the background for a pleasant, stimulating, and long-lasting avocation. Begun so long ago, the reading of "Hamlet" yearly has been a source of continuous joy. Dover Wilson, in addition, was linked as a warm friend by the bookish bridge and by correspondence. In spite of all that I once must have known and subsequently largely forgotten about economics, physics, social

science, general history, and other forms of literature (acquired in sufficient quantity at least to have earned a degree at Dartmouth in 1912) there apparently remained a small core of selective curiosity, sufficient to keep the mind open. When the opportunity came the serviceable association habits were ready to grasp it. To develop such habits should be the fundamental aim of a college of Liberal Arts. If you do not carry that type of equipment across the bridge from college to medical school, you cannot expect to have a satisfactory career in medicine.

Even before the association with Dover Wilson, however, I had by chance met Robert Bridges, the poet and former house physician at St. Bartholomew's Hospital in London. I was in Oxford when the "Testament of Beauty" was published in October, 1929. Catching up the first edition quickly, I soon found that Bridges had thrown a new and brilliant spotlight on the character of Shakespeare's Hamlet. "Hamlet himself would never had been aught to us, or we to Hamlet, wer't not for the artful balance whereby Shakespeare so gingerly put his sanity in doubt without the while confounding his Reason."

That "artful balance," so gingerly put on, gives a workable key to the enigma of the most elusive figure in all literature, and makes Hamlet "aught to us" and "we to Hamlet" as never before.[5]

Now there is, too, an artful balance between the scientific aspects of medicine and the cultural standards of the art. Without a knowledge of medical history and the significance of the accomplishments of our predecessors it is nearly impossible to maintain our equilibrium in a field of endeavor such as medicine. We must weigh carefully and gingerly the art against the science. Medical history offers a tool to balance the scale, and indeed without it science becomes, except for a few, an intolerable burden. The tool is easily acquired, for every medical student, as soon as he enters the door of his school, encounters numerous clues to the history of his subject. Medicine is replete with eponyms, and in the first weeks of anatomy he meets some of the greatest names in all medicine. Scarpa's triangle, the Fallopian tubes, the sphincter of Oddi, the Rolandic fissure, the island of Reil, and many others stimulate your curiosity in the new world of the fabric of the body and in those who first described it. Poor would be the student whose bump of inquisitiveness was not aroused by these eponymic designations. After reaching the stage of clinical medicine you will meet another galaxy of names, such as Addison's disease, Parkinsonism, Bright's disease, Bell's palsy, Cheyne-Stokes respiration, Arnold-Chiari's malformation, Hand-Schüller-Christian disease, and many more.[6] This is "entering medical history through the back door," as Fielding Garrison once remarked, and perhaps it is the best and easiest way to become acquainted with the past on which we must base so much of our productive thinking of the future. Without a knowledge of what has gone before, the foreseeable future is dim indeed, for each of you stands on the shoulders of those who have passed the same way, and outstanding physicians who have not taken a profound interest in the history of medicine are almost unknown.

Indeed if you have not absorbed in your academic career those principles which I have mentioned, you will find it difficult to satisfy yourself in the pure practice of a profession without also constantly seeking means to nourish the cultural aspects of life. Encompassed as you will be with all the complexities of your medical career, if you do not step beyond the narrow bounds of hospital and practice, you will soon lose the art of a balanced existence that gives a stability impossible to achieve on the narrow tightrope of specialization. All great physicians have felt this, and perhaps I can give you one or two examples from the lives of men I have known in my brief lifetime to illustrate the importance of linking humanism, history, and medicine.

None was greater than Sir William Osler[7] whom I had the privilege of living with during a year in Oxford, after I had

graduated from the Harvard Medical School. He never approached a patient without thinking in terms of history, balancing the pros and cons of diagnosis on the basis of observation, combined with a historical evaluation of their importance. Whenever he faced a problem of diagnosis and particularly prognosis, one could almost hear him say to himself, "How would Hippocrates have looked at this situation, or Harvey, or Vesalius, or Sydenham?" When Harvey Cushing sent me abroad he told me what he considered the most important thing for me to look for in Osler's practice. "Notice how he approaches the patient," he said, "but even more important how he leaves the patient." Of course what he had in mind was that I could absorb the fundamentals of diagnosis, pathology, and treatment in the hospital, but from Osler I must look for something more, for he carried with him in a supreme degree the art of practice as well as the science. No one, moreover, knew medical history better than Osler. His library, of course, was an inspiration to me and set me on a path from which I have never deviated in the more than forty years since I was Osler's pupil.[8]

Harvey Cushing,[9] too, was an excellent example of the balance between the art and practice of medicine. Developing a highly technical specialty of his own and becoming world famous in a field pioneered by his efforts, he, nevertheless, was a lifelong student of medical history. Perhaps he will longest be remembered for his final book, "A Bio-Bibliography of Andreas Vesalius," [10] published after his death, the culmination of an interest stimulated in Cushing by Osler that dates from 1898 or possibly even earlier. As John Fulton says in the preface, "In the impetuous Vesalius, Harvey Cushing saw a vigorous and indefatigable character with a stormy temperament similar to his own, a man with artistic leanings who gave unremitting attention to detail, all of which aroused Cushing's admiration and caused him to follow Vesalius with instinctive determination and persistence." Cushing pursued the trail for more than forty years, collecting everything conceivable that he could relating to Vesalius, and spending some of his summer vacations traveling through Europe uncovering new facts concerning the man and his works. On one of those occasions I accompanied him, and I shall always be inspired by the enthusiasm that Cushing threw into his historical work as an avocation. His pace could only be compared in intensity with a similar persistence in his surgical research and the unfolding of a strict ritual of operation. Here again was a great man, balancing one against the other to complete the whole. Cushing and Osler are perhaps the outstanding examples of the intertwining of surgery, medicine, and cultural attainments, using familiar tools carried over from their academic years into their professional life.

There are a few more examples of this linkage of culture and medicine worthy of bringing to your attention. One is a distinguished orthopedic surgeon of Bologna, Vittorio Putti,[11] whose hospital and practice set a standard of orthopedics in Italy that has not, so far as I am aware, been equalled in that country. Like Cushing, Putti was a dynamic surgeon and enthusiastic pioneer, and in addition a great lover of the history of his specialty. We visited him one summer and absorbed some of the scholarly atmosphere that surrounded everything that he did. He took us on a trip to Cortina in the Dolomites, where he had a summer hospital for patients with bone tuberculosis. In the beautiful hills of northern Italy he had built an institution which was outstanding of its kind. But equally impressive was his work in medical history culminating in a life of Berengario da Carpi [12] which is almost unequalled as a biography of a medical figure. This work was his secondary occupation over a period of many years and added greatly to his stature as a physician. From Berengario, as Cushing had done from Vesalius, Putti drew inspiration, for he stands out as a towering figure, clean and straight as the Dolomites he loved so well, as did his predecessor in Bologna.

Equally outstanding is a man, fortunately still living, of quite another stamp, a thoracic surgeon of London who has become one of the leading bibliographers of all time. I refer to Geoffrey Keynes,[13] whose bibliographies have led him to the presidency of the Bibliographical Society of London. Few works have been more meticulously written, and indeed they are superb examples of a particular branch of literature not widely developed outside of Great Britain. Some of his books have gone into second or even third editions,[14] a situation almost unknown before Geoffrey Keynes pioneered in this art. You will become familiar as time goes on with his bibliography of William Harvey [15] and of Sir Thomas Browne,[16] as well as many others, for he put into these works the same enthusiasm and creative writing that Cushing put in his Vesalius, or Putti in his Berengario da Carpi. Each of these four men accomplished all of this cultural attainment while at the same time leading their respective fields of medicine and surgery and setting examples of hard, unremitting labor, resulting in rich rewards for those that have come after them.

Unique in the field of medicine and literary attainments is J. Johnston Abraham, a retired London surgeon, author, soldier, traveler, novelist, playwright, and medical advisor to one of the great publishing houses in England. His recent autobiography [17] is full of interest and should be read by all medical students. Born in northern Ireland, he trained in Dublin and went to London for his internship. While wating for a residency he took a trip as the ship's doctor on a freight vessel carrying a few passengers. The vessel touched at various Mediterranean countries and ultimately went to the Far East. On his return he wrote up his diary and, as it seemed to interest a good many of his friends, decided to send it to a publisher. On nine occasions it was rejected, sometimes after a few weeks and once as long as six months. Naturally discouraged, any man of less stature would have torn up the manuscript and thrown it in the waste basket. Happening by chance, however, to see the name of the publishing firm of Chapman and Hall, he made one last desperate effort, retyped the dog-eared title page, and sent it in to one of the most renowned publishing firms in London, who had published the works of Charles Dickens. Much to his surprise it was accepted, published [18] in 1911, and became the most widely read and popular travel book ever written by a doctor. In 1949 when I visited Abraham in London he presented me with a copy of a new edition. The total printing at that time had reached half a million, and the book still sells at the rate of several thousand a year.

This, of course, brought immediate fame in the literary world to Johnston Abraham, but because he was entering the field of surgery he felt it best to suppress his name, as it was not considered good form for a surgeon to have such a profitable avocation in addition to his regular career. He therefore published a number of books under other names and one of them was made into a successful motion picture. His finest piece of writing, however, was undoubtedly his life of John Coakley Lettsom,[19] the Quaker physician in London who played such a role in the development of medical education in this country that he may be considered as the father of two physicians, both pioneers in medical education. Benjamin Waterhouse studied with Lettsom and returned to this country in 1782 to become the first Professor of the Theory and Practice of Medicine in the newly established Harvard Medical School. Nathan Smith, the founder of Dartmouth Medical School as well as the schools at Yale, Bowdoin, and Vermont, and who had graduated from Harvard Medical School in the third class in 1790, carried letters of introduction from Waterhouse and John Warren of Boston to Lettsom when he went to London in 1796. You will remember that he had borrowed money from his friend to make the long journey, confident that the Trustees of Dartmouth would establish a medical school. In England he bought the necessary books and instruments, and in collecting these he found Lettsom's

advice and help useful. Lettsom also contributed generously from his own library and assisted him to pay for the apparatus for the anatomical, surgical, and chemical sections of the proposed school. Returning to Boston in the autumn of 1797, Smith soon started his lectures in Hanover and at first occupied the chairs of anatomy, surgery, chemistry, theory and practice of physic; "a whole settee of professorships," according to Oliver Wendell Holmes.

This book by Abraham is certainly his chief source of literary fame and indeed may be the main source of his memory in future years.

The list might be greatly expanded by adding the names of D'Arcy Power, Henry Head, Charles Sherrington, Eric Waller, and other teachers and friends, but I must leave to you the rewarding pleasures of making their acquaintance as you grow in stature by further exploration.

But perhaps you will consider it "too curiously, to consider so." On the soundness of planning such a course for a career in medicine or surgery there may be brought to your mind some indecision, some doubts wrestling with convictions, a little "oscillation on the pavement," as Sherlock Holmes would have said. Are these men the dilettantes of the profession? Not if I read the story correctly. Everyone must seek to escape, at least in part, the berattled atmosphere of the modern world, with science turning the wheel in a push-button age. Such an existence gives us scant time for those inspired moments so precious if we are to do a man's work in a manner that will give us a full life. Satisfaction can come only if ear and mind take the full flood of overtones and associations directly, freely, and easily, as a nook of the bay takes the spring tide. It is those overtones and associations that we should carry over from our educational tour in a college of Liberal Arts into the restricted field of scientific medicine. Time must be allowed to take our own mental and spiritual pulse, and humanism and medical history give us a point of view so that we are not standing up too close or back too far. In spite of conventional entanglements one needs in this age of transition, as in all times past, a broad feeling for life and a spacious and generous understanding of it. All great physicians have had this and, on a basis of cultural and humanistic standards developed through a study of the past, they have penetrated into the lives of our forebears for inspirations that have directed their own careers.

In all our endeavors the rule giving the most gratification and contentment is the Biblical counsel, "Hold fast to that which is good." In spite of the grammarians' quibble, "that which is good" has a wholesome and satisfying meaning, but "hold fast" is an admonitory warning hardly needed for a graduate of Dartmouth College.

8 The Fenway (15).

NOTES

1. The edition of the play used by me in college was edited by William J. Rolfe, a high school teacher, first published in 1878 and only moderately revised in 1909. The analysis of the character of Hamlet by Rolfe was based on sound reasoning, but the notes were almost entirely lacking in any adequate discussion of the dramatic significance of each word and action of the play as disclosed by Shakespeare in the progressive unfolding of the plot. Writing for elementary students in the Victorian era, Rolfe was careful to omit comment on Hamlet's quibbles, particularly if they implied an indelicate suggestion, thus eliminating one of the main roots of Shakespeare's poetic expression, for the author of Hamlet habitually thought in terms of quibbles.

2. Hamlet, edited by John Dover Wilson, London, Cambridge University Press, 1934. (Second edition, 1936; reprinted, 1954, with slight editorial changes.)

3. The Hamlet text of "The New Cambridge," as this series of plays is now known, is based on a meticulous study of the quarto (1605) and folio (1623) editions (Wilson, J. D.: The Manuscript of Shakespeare's Hamlet and the Problems of Its Transmission, 2 volumes, London, Cambridge University Press, 1934). These volumes are superb examples of modern literary detection in critical bibliography.

4. Wilson, J. D.: What Happens in Hamlet, London, Cambridge University Press, 1935. (Second Edition, 1937; Third Edition, 1951.) Books published by the Cambridge University Press in England are obtainable at the American Branch, New York.

5. Other than the outstanding Cambridge University Press edition of Hamlet, the most readable and up-to-date is The Pelican Shakespeare, edited by Willard Farnham, Baltimore, Penguin Books, 1957. The Folger Library General Reader's Shakespeare, New York, Pocket Library, 1958, seems to me less satisfactory. Each is issued in inexpensive, paper-back form.

6. A sound medical dictionary is one of the first books a medical student should acquire. You will wear out a good many successive editions in your lifetime, in whatever branch of medicine you make your career. Three excellent dictionaries are currently available: Blakiston's New Gould Medical Dictionary, Second Edition, New York, The Blakiston Division, McGraw-Hill Book Company, Inc., 1956; Dorland's Illustrated Medical Dictionary, 23d Edition, Philadelphia, W. B. Saunders Company, 1957; Stedman's Medical Dictionary, 19th Revised Edition, Baltimore, The Williams & Wilkins Company, 1957. Like books on anatomy, dictionaries are plentiful and admirable; use makes all the difference.

7. Cushing, H.: The Life of Sir William Osler. Two volumes, Oxford, Clarendon Press, 1925. Reprinted in one volume, London, Oxford University Press, 1940. One of the outstanding medical biographies of all time. The details at first may seem a little overwhelming, but the spirit of Osler is best read in his letters and occasional remarks. Cushing has caught the blitheness of the man as well as the greatness of his heart and mind.

8. Viets, H. R.: A Roving Commission: The Doctor Calls on Some of His Friends, Bull. Hist. Med. *22*:363-372, 1948. A brief account of Osler at Oxford and of Arnold Klebs at Nyon, Switzerland. The home of Klebs, a retired American physician, on Lake Leman, was the center of much activity, long a rallying point for summer vacationists with medicohistorical leanings.

9. Fulton, J. F.: Harvey Cushing: A Biography, Springfield, Ill., Charles C Thomas, Publisher, 1946. A reviewer commented in 1947: "Modestly priced to fit the pocket of the medical student of the day, the book should be compulsory reading for every man and woman entering medicine as a profession. Poor indeed is the student who cannot gain from reading the life of one of America's finest products, a man who inspired his contemporaries as few others have done and who will continue to enrich the lives of those that come after him, thanks to this worthy volume."

10. Cushing, H.: A Bio-Bibliography of Andreas Vesalius, New York, Henry Schuman, Inc., 1943.

11. Viets, H. R., and Osgood, R. B.: Vittorio Putti, New England J. Med. *223*:955-6, 1940.

12. Putti, V.: Berengario da Carpi: Saggio biografico e bibliografico sequito dalla traduzione del "De fractura calvae sive cranei," Bologna, L. Cappelli, 1937.

13. Fulton, J. F.: The Great Medical Bibliographers: A Study in Humanism, Philadelphia, University of Pennsylvania Press, 1951.

14. Keynes, G.: A Bibliography of John Donne, Cambridge University Press, 1914. (Second Edition, 1932; Third Edition, 1958.)

15. Keynes, G.: A Bibliography of the Writing of Dr. William Harvey, 1578-1657, London, Cambridge University Press, 1928. (Second Edition, revised, 1953.) A few copies of the second edition were printed with the incorrect date of Harvey's birth (1573 for 1578) on the title page and dust wrapper.

16. Keynes, G.: A Bibliography of Sir Thomas Browne, Kt., M.D., London, Cambridge University Press, 1924.

17. Abraham, J. J.: Surgeon's Journey: The Autobiography of J. Johnston Abraham, London, William Heinemann, Ltd., 1957.

18. Abraham, J. J.: The Surgeon's Log, Being Impressions of the Far East, London, Chapman & Hall, Ltd., 1911. (Revised edition with new preface and additional chapter, 1947.)

19. Abraham, J. J.: Lettsom: His Life, Times, Friends and Descendants, London, William Heinemann, Ltd., 1933.

Intestinal Obstruction Produced by Fecalith Arising in Meckel's Diverticulum

RICHARD J. FIELD SR., M.D., and RICHARD J. FIELD JR., M.D., Centreville, Miss.

The history of Meckel's diverticulum and its complications is dominated by the unusual. Not often is one able to make a diagnosis prior to surgery, and the element of surprise is quite evident on opening the peritoneal cavity. Such were the circumstances in the following case report.

Report of a Case

A 52-year-old white man, a pharmacist, was admitted to the Field Memorial Community Hospital on Nov. 1, 1957, with a diagnosis of acute alcoholism. The patient had been drinking for approximately 14 days and had reached a point requiring medical care. Thirty-six hours after admission the patient was rational, retaining liquids and apparently on the road to recovery, when he complained of cramping abdominal pains and vomited once. These generalized cramping abdominal pains persisted, without further nausea or vomiting. The pains were not excruciating, but uncomfortable. There was no flatus following the initial episode. Six hours after the onset of these abdominal pains there was noticeable symmetric distention of the abdomen with increased resonance to percussion and high-pitched peristaltic sounds on auscultation. Rectal examination showed the rectum to be empty, but otherwise within normal limits.

Eight hours following the onset of cramping abdominal pain, flat and erect films of the abdomen were made, which showed rather marked distention of the small bowel, with absence of gas in the large bowel. There were fluid levels in the small bowel on the erect film.

With the above history and roentgenologic examination, a diagnosis of distal small-bowel obstruction was made and the patient prepared for surgery.

The abdomen was opened and obvious distention of the small bowel was immediately revealed (Fig. 1). A golf-ball-sized mass was palpated approximately 5 cm. above the ileocecal junction, which was rather firm and completely blocked the ileum at this point. Further exploration revealed the presence of the large Meckel's diverticulum. The diagnosis was then obvious, and the fecalith was

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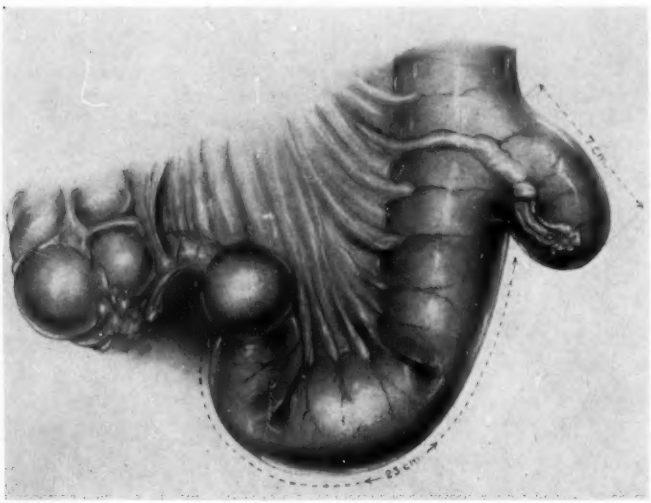

Fig. 1. — Abdominal findings at the time of exploration. Appendix removed previously.

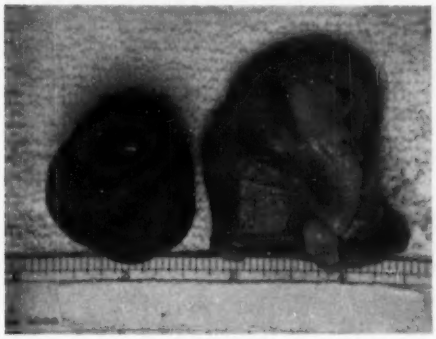

Fig. 2.—Diverticulum and the fecalith arising within it, taken immediately after surgery. Rule calibrated in inches.

manipulated back into its bed in the Meckel's diverticulum. The diverticulum was then removed by excising its base transversely and closing the defect with interrupted cotton sutures, as described by Gross.[6]

The patient gave a history of recurrent upper abdominal pain, and on several occasions upper gastrointestinal radiographs were made, with a possible diagnosis of duodenal ulcer. They were always reported as within normal limits. Interestingly enough, since surgery these pains have completely disappeared. The pathological report showed only small-bowel mucosa lining the diverticulum, and no evidence of ectopic gastric or pancreatic tissue.

Comment

In this case the fecalith had apparently been developing over many years and was the source of the patient's vague abdominal pains. It is assumed that the increased peristalsis with his acute alcoholism caused the extrusion of the fecolith from the diverticulum. A careful search of the surgical literature leads us to believe that this is the second reported case of obstruction by a fecalith from Meckel's diverticulum in the American surgical literature. The first was reported by Danzis[3] in 1950. There have been many reports of the presence of concretions and calculi in the excised Meckel's diverticulum, but only in the one other case was the fecalith large enough to produce obstruction of the small bowel. The diverticulum undoubtedly has peristaltic activity, although to a much less extent than the small bowel; otherwise, one would suspect that the stagnation of fecal contents within the diverticulum would produce these large fecaliths much more frequently.

Summary

A patient with a small-bowel obstruction due to a large fecalith which arose in Meckel's diverticulum is presented. The fecolith extruded from the diverticulum, owing to hyperperistalsis following an acute alcoholic episode.

The Field Clinic.

REFERENCES

1. Allen, A. W., and Donaldson, G. A.: Meckel's Diverticulum Containing Calculi, Arch. Surg. 50: 286-287 (June) 1945.

2. Appell, A. A., and Klotz, P. G.: Meckel's Diverticulum Containing Calculi, Canad. M. A. J. 66:476-478 (May) 1952.

3. Danzis, M.: Intestinal Obstruction in Meckel's Diverticulum with Report of a Case with a Large Enterolith, J. M. Soc. New Jersey 47:548-553 (Dec.) 1950.

4. Gile, J. F., and MacCarthy, W. C., Jr.: Calcified Concretions Within a Meckel's Diverticulum, Radiology 41:491-494, 1943.

5. Gross, R. E.: Surgery of Infancy and Childhood, Philadelphia, W. B. Saunders Company, 1953.

6. Kiesewetter, W. B.: Meckel's Diverticulum in Children, A. M. A. Arch. Surg. 75:914-919 (Dec.) 1957.

7. Michel, M. L.; Field, R. J., and Ogden, W. W., Jr.: Meckel's Diverticulum: An Analysis of 100 Cases and the Report of a Giant Diverticulum and of 4 Cases Occurring Within the Same Family, Ann. Surg. 141:819-826 (June) 1955.

8. Mulsow, F. W.: Meckel's Diverticulum Containing Calculi: Case Report, Am. J. Digest. Dis. 10:188-189, 1943.

9. Sherren, J.: Meckel's Diverticulum Containing Calculi and Producing Colic, Proc. Roy. Soc. Med. (Clin. Sect.) 3:11-15, 1909-1910.

Further Studies on the Question of an Inhibitory Hormone from the Gastric Antrum

LESTER R. DRAGSTEDT, M.D., Ph.D.; SOICHI KOHATZU, M.D.; JAMES GWALTNEY, M.D.; KAZUO NAGANO, M.D., and HERBERT B. GREENLEE, M.D., Chicago
With the Technical Assistance of Donald B. Thursh and Richard A. Weinberg

According to Babkin,[1] Sokolov, working in Pavlov's laboratory, discovered that in the dog the introduction of 0.5% of HCl into the stomach or of gastric juice into the duodenum markedly diminished the secretion of gastric juice from a Pavlov pouch. These observations were confirmed and extended by Wilhelmj and his associates[2-4] and led these authors to formulate the theory that when the gastric contents become sufficiently acid in reaction, the gastric phase of secretion is inhibited. This theory was strongly supported by experimental studies in this laboratory reported by Oberhelman, Woodward, Zubiran, and Dragstedt[5] and by Woodward, Lyon, Landor, and Dragstedt.[6] In dogs prepared with both the Heidenhain pouch and an isolated pouch of the antrum, the introduction of various foods into the isolated antrum provoked a vigorous secretion of gastric juice from the Heidenhain pouch. Distention of the isolated antrum also produced a copious secretion of gastric juice from the fundic pouch. These secretory responses were obtained only when the test food was neutral or faintly acid in reaction, or when the distention was produced by balloons or by neutral fluids. If the pH of the food mixture was lower than 2.5-5.0 or if the fluid used for distention of the antrum pouch was acid in reaction, no stimulation of secretion from the Heidenhain pouch occurred. Furthermore, when the antrum of the stomach together with a portion of the corpus was transplanted as a diverticulum into the colon, a vigorous stimulation of gastric secretion resulted. This stimulation was markedly increased when the acid-secreting mucosa was excised from the transplanted antrum pouch. Perfusion of the isolated antrum pouch with acid, however, did not inhibit the secretion of gastric juice produced by insulin hypoglycemia, or by the injection of histamine. These findings led to the conclusion that perfusion of the antrum with acid inhibited the antral phase of gastric secretion, probably owing to direct interference with gastrin formation or release.

The possibility of an inhibitory hormone from the antrum mucosa being responsible for this phenomenon was considered, especially in view of the demonstration by Brunschwig and his associates[7] and by Hood, Code, and Grindlay[8] of a gastric-secretory depressant in gastric juice. Harrison, Lakey, and Hyde[9] have reported a series of interesting experiments which they interpret as demonstrating that the antrum under certain conditions produces a substance which actively inhibits the secretion of acid by the stomach. In dogs prepared with Heidenhain pouches the secretion of gastric juice was augmented by the transplantation of the distal half of the gastric antrum into the colon as a diverticulum by the method of Dragstedt, Woodward, Oberhelman, Storer, and Smith.[10] Subsequent removal of the proximal portion of the gastric antrum from the stomach caused still further increase in gastric secretion

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From the Department of Surgery of The University of Chicago.

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from the Heidenhain pouch. It was concluded that the portion of antrum remaining in situ in the stomach was exerting an inhibitory influence on the secretion from the Heidenhain pouch, as demonstrated by the marked increase which occurred when this portion of the antrum was excised.

In a previous study from this laboratory,[11] this question of an inhibitory hormone from the gastric antrum was examined, and experiments were performed which appeared decisive, to the effect that acid in contact with the antrum mucosa does not release an inhibitory hormone with any significant potency. Animals were prepared with a vagus-denervated Heidenhain pouch and two isolated pouches of the antrum. The introduction of food or acetylcholine solutions into either of the antrum pouches produced strong stimulation of gastric secretion from the Heidenhain pouch. When, however, the Heidenhain pouch was being stimulated to secrete by the introduction of food into one of the antrum pouches, the introduction of acid solutions into the other antrum pouch produced no significant inhibition of secretion. Such inhibition would have been expected if a potent inhibitory hormone were released from the antrum mucosa on contact with acid. In view of these negative findings, we repeated the experiment of Harrison and his associates with certain modifications and extensions, which provide an alternative interpretation to the one supplied by these authors.

Experimental Procedure

Four dogs were prepared for the experiment as described by Harrison, Lakey, and Hyde. The animals were operated upon under ether anesthesia and with the usual aseptic precautions. A Heidenhain pouch was prepared with a stainless-steel cannula for the quantitative collection of gastric juice by the method previously described from this laboratory. The distal half of the antrum was transplanted into the transverse colon as a diverticulum. After the animals had recovered from the operation and were eating the stock diet of the laboratory, quantitative 24-hour collections of the gastric juice secreted by the Heidenhain pouch were made. The volume was measured and the free-acid concentration titrated with Toepfer's reagent. The 24-hour acid output, in milliequivalents, was calculated by multiplying the volume, expressed in liters, by the free-acid concentration, expressed in clinical units. After a minimum of 30 daily collections of gastric juice had been made, the proximal portion of the antrum remaining in continuity with the fundus was excised. Daily collections of gastric secretion were then continued from 40 to 50 days, after which the antrum transplant in the colon was removed. The data secured are graphically illustrated in Figures 1 to 4. There is a slight increase in gastric secretion in Figure 2, no increase in Figures 1 and 3, but a rather marked increase in Figure 4. In each instance, removal of the antrum transplant from the colon practically abolished the secretion of gastric juice from the Heidenhain pouch.

Because of the lack of uniformity in these data, the experiment was continued with four additional animals, D-267, D-302, D-303, and D-316. The experiment was modified only to the extent of measuring the secretion from the Heidenhain pouch during a control period, when the entire antrum was in its normal location. Somewhat to our surprise, in the first two experiments illustrated in Figures 5 and 6, transplantation of the distal half of the antrum into the colon as a diverticulum resulted in a decrease in the secretion of gastric juice from the Heidenhain pouch, instead of the expected increase. After removal of the residual antrum left in continuity with the fundus, the secretion increased slightly in Animal D-267 (Fig. 5) but decreased slightly in Animal D-302 (Fig. 6). In both Animal D-303 (Fig. 7) and Animal D-316 (Fig. 8) gastric secretion from the Heidenhain pouch increased materially above the control level when the distal half of the antrum was transplanted into the colon as a

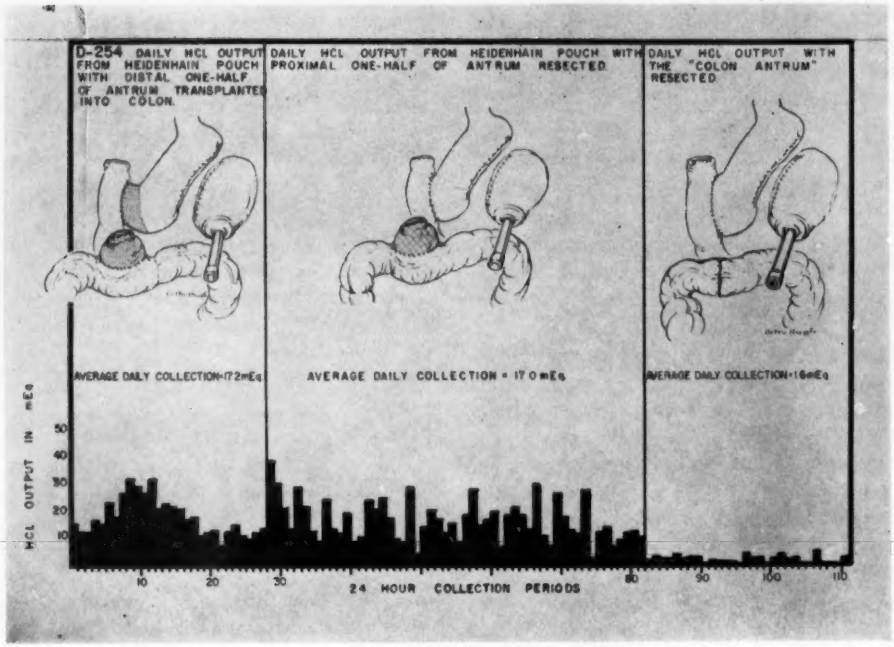
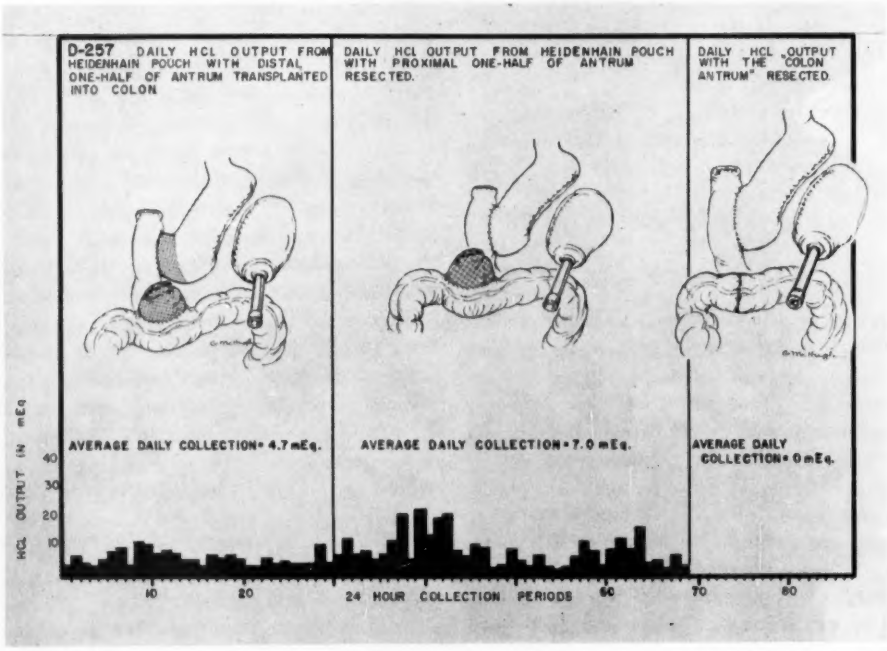


Fig. 1.—Dog D-254

Fig. 2.—D-257



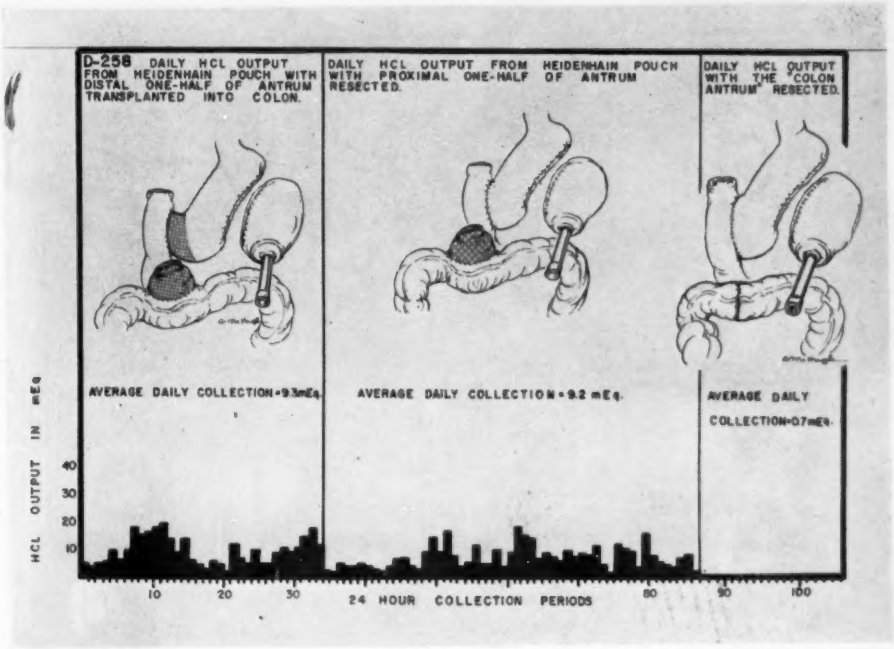
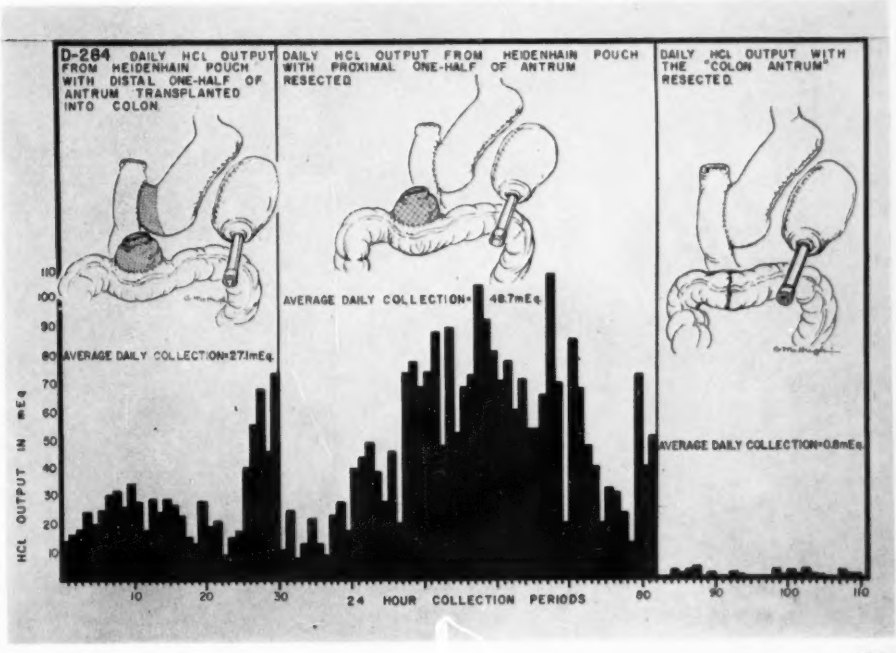


Fig. 3.—D-258

Fig. 4.—D-284



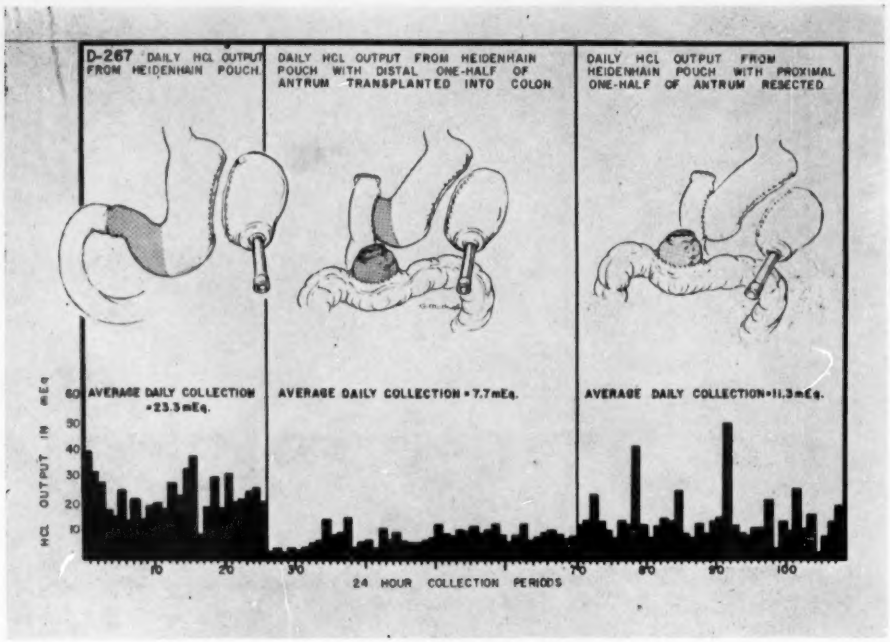
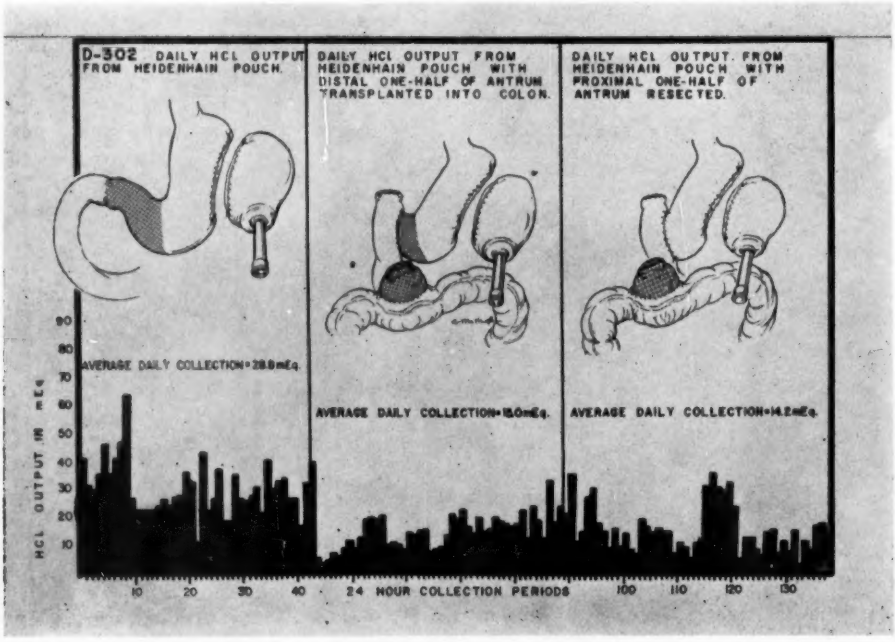

D-267 DAILY HCL OUTPUT FROM HEIDENHAIN POUCH.
DAILY HCL OUTPUT FROM HEIDENHAIN POUCH WITH DISTAL ONE-HALF OF ANTRUM TRANSPLANTED INTO COLON.
DAILY HCL OUTPUT FROM HEIDENHAIN POUCH WITH PROXIMAL ONE-HALF OF ANTRUM RESECTED
AVERAGE DAILY COLLECTION =23.3mEq.
AVERAGE DAILY COLLECTION = 7.7mEq.
AVERAGE DAILY COLLECTION=11.3mEq.
HCL OUTPUT IN mEq
24 HOUR COLLECTION PERIODS

Fig. 5.—D-267

Fig. 6.—D-302


D-302 DAILY HCL OUTPUT FROM HEIDENHAIN POUCH.
DAILY HCL OUTPUT FROM HEIDENHAIN POUCH WITH DISTAL ONE-HALF OF ANTRUM TRANSPLANTED INTO COLON.
DAILY HCL OUTPUT FROM HEIDENHAIN POUCH WITH PROXIMAL ONE-HALF OF ANTRUM RESECTED.
AVERAGE DAILY COLLECTION=28.8mEq.
AVERAGE DAILY COLLECTION=15.0mEq.
AVERAGE DAILY COLLECTION=14.2mEq.
HCL OUTPUT IN mEq
24 HOUR COLLECTION PERIODS

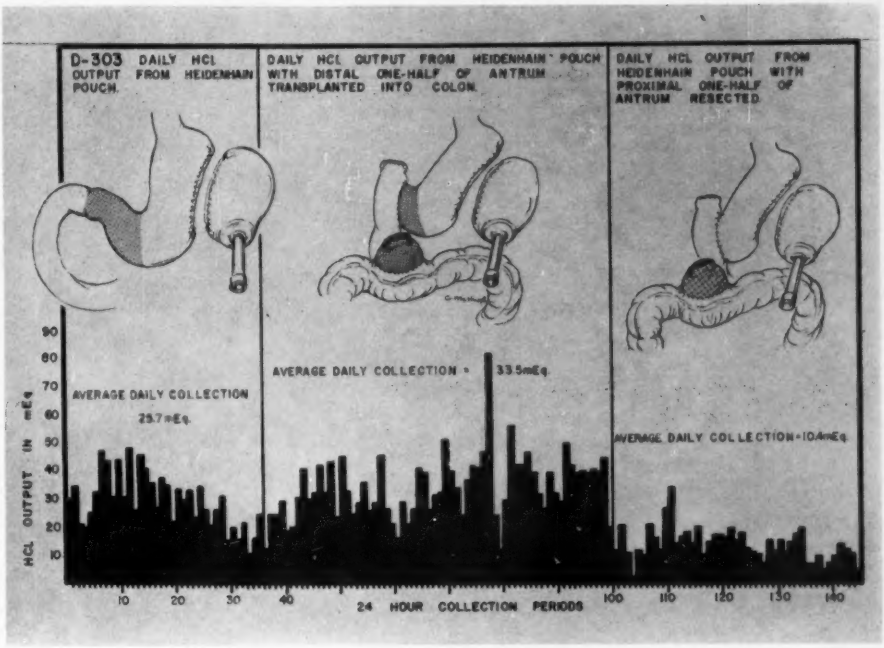
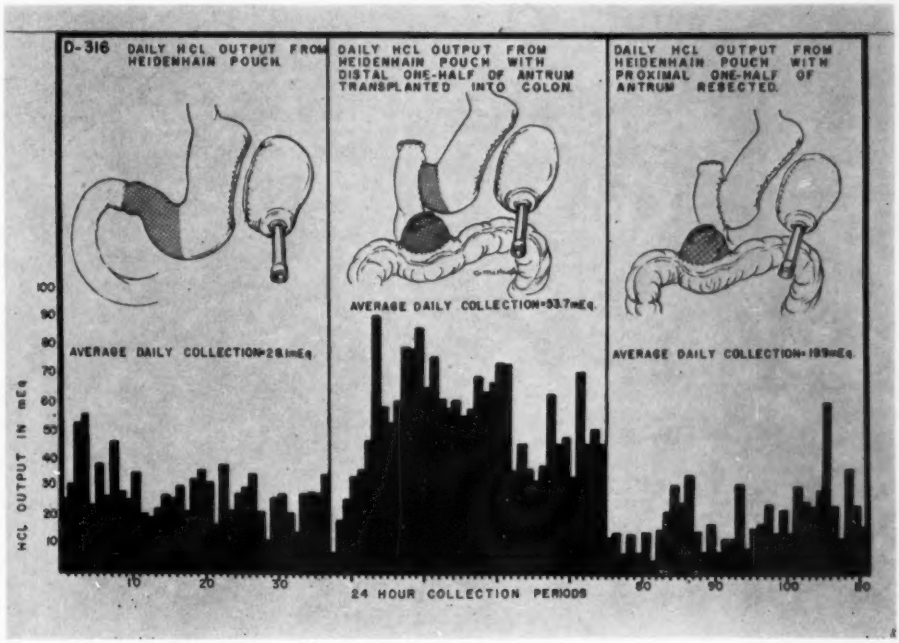

D-303 DAILY HCL OUTPUT FROM HEIDENHAIN POUCH.
DAILY HCL OUTPUT FROM HEIDENHAIN POUCH WITH DISTAL ONE-HALF OF ANTRUM TRANSPLANTED INTO COLON.
DAILY HCL OUTPUT FROM HEIDENHAIN POUCH WITH PROXIMAL ONE-HALF OF ANTRUM RESECTED.
AVERAGE DAILY COLLECTION 25.7 mEq.
AVERAGE DAILY COLLECTION = 33.5 mEq.
AVERAGE DAILY COLLECTION = 10.4 mEq.
HCL OUTPUT IN mEq.
24 HOUR COLLECTION PERIODS

Fig. 7.—D-303

Fig. 8.—D-316


D-316 DAILY HCL OUTPUT FROM HEIDENHAIN POUCH.
DAILY HCL OUTPUT FROM HEIDENHAIN POUCH WITH DISTAL ONE-HALF OF ANTRUM TRANSPLANTED INTO COLON.
DAILY HCL OUTPUT FROM HEIDENHAIN POUCH WITH PROXIMAL ONE-HALF OF ANTRUM RESECTED.
AVERAGE DAILY COLLECTION = 28.1 mEq.
AVERAGE DAILY COLLECTION = 33.7 mEq.
AVERAGE DAILY COLLECTION = 19.9 mEq.
HCL OUTPUT IN mEq.
24 HOUR COLLECTION PERIODS

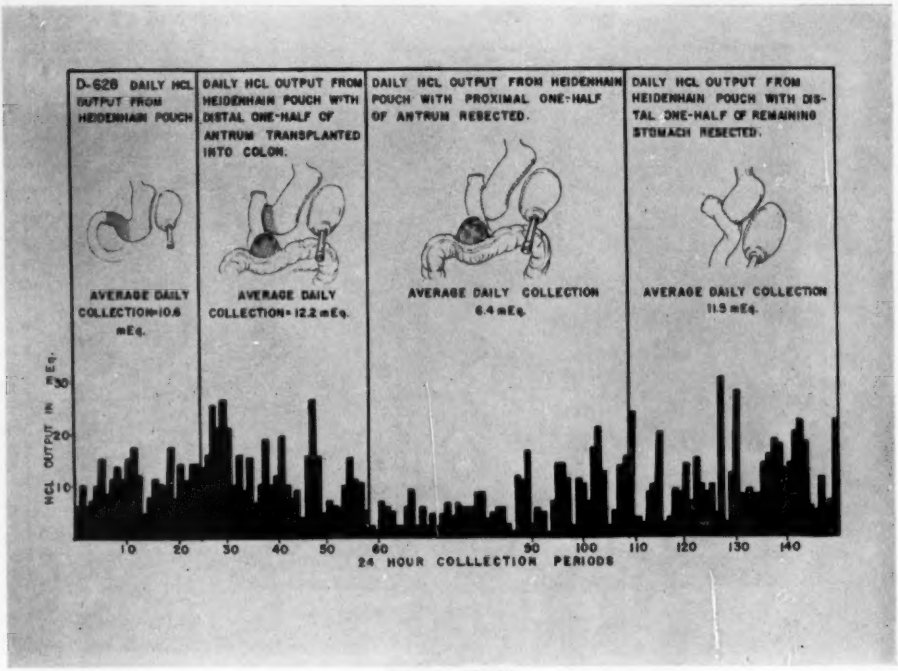
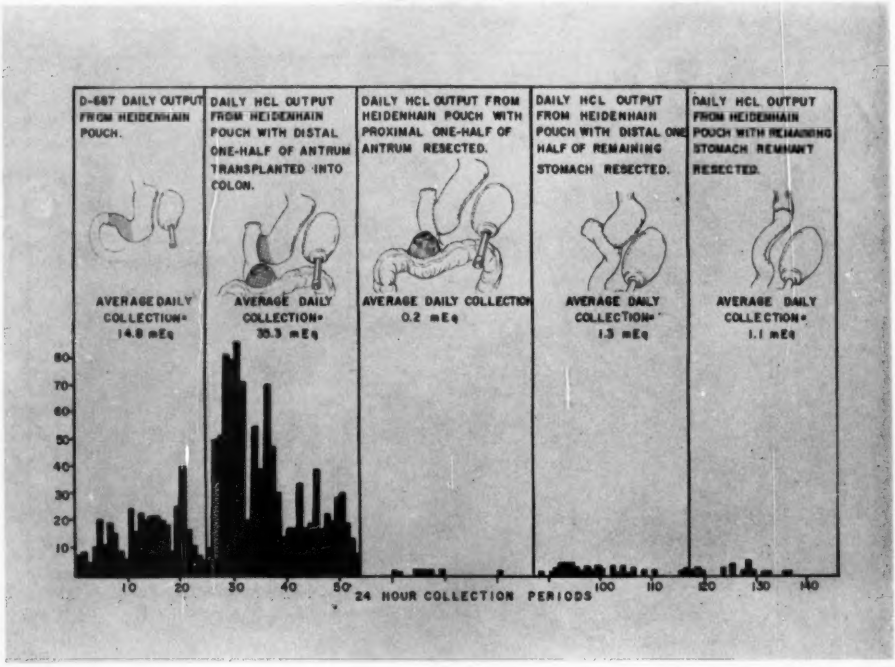

D-628 DAILY HCL OUTPUT FROM HEIDENHAIN POUCH
AVERAGE DAILY COLLECTION=10.6 mEq.
DAILY HCL OUTPUT FROM HEIDENHAIN POUCH WITH DISTAL ONE-HALF OF ANTRUM TRANSPLANTED INTO COLON.
AVERAGE DAILY COLLECTION= 12.2 mEq.
DAILY HCL OUTPUT FROM HEIDENHAIN POUCH WITH PROXIMAL ONE-HALF OF ANTRUM RESECTED.
AVERAGE DAILY COLLECTION 6.4 mEq.
DAILY HCL OUTPUT FROM HEIDENHAIN POUCH WITH DISTAL ONE-HALF OF REMAINING STOMACH RESECTED.
AVERAGE DAILY COLLECTION 11.9 mEq.
HCL OUTPUT IN mEq.
10
20
30
10 20 30 40 50 60 90 100 110 120 130 140
24 HOUR COLLLECTION PERIODS

Fig. 9.—D-628

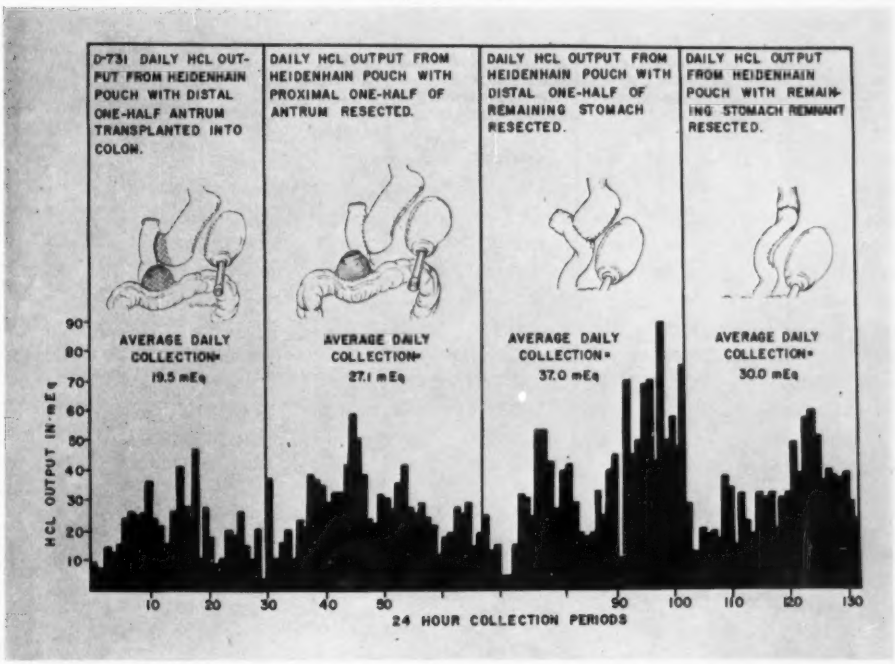
Fig. 10.—D-687


D-687 DAILY OUTPUT FROM HEIDENHAIN POUCH.
AVERAGE DAILY COLLECTION= 14.8 mEq
DAILY HCL OUTPUT FROM HEIDENHAIN POUCH WITH DISTAL ONE-HALF OF ANTRUM TRANSPLANTED INTO COLON.
AVERAGE DAILY COLLECTION= 35.3 mEq
DAILY HCL OUTPUT FROM HEIDENHAIN POUCH WITH PROXIMAL ONE-HALF OF ANTRUM RESECTED.
AVERAGE DAILY COLLECTION 0.2 mEq
DAILY HCL OUTPUT FROM HEIDENHAIN POUCH WITH DISTAL ONE HALF OF REMAINING STOMACH RESECTED.
AVERAGE DAILY COLLECTION= 1.3 mEq
DAILY HCL OUTPUT FROM HEIDENHAIN POUCH WITH REMAINING STOMACH REMNANT RESECTED.
AVERAGE DAILY COLLECTION= 1.1 mEq
80
70
60
50
40
30
20
10
10 20 30 40 50 100 110 120 130 140
24 HOUR COLLECTION PERIODS

diverticulum. However, in both these animals, removal of the residual antrum from the stomach caused a marked fall in the output of gastric juice rather than an increase, such as that reported by Harrison and his associates.

These findings, contradictory to those of Harrison, Lakey, and Hyde, induced us to repeat the experiment of five additional animals with a modification which, it was hoped, would throw light on the cause of our conflicting findings. The border between the antrum and the fundus, or corpus, of the stomach is not sharply defined, and it occurred to us that when the residual antrum left in connection with the corpus was excised, a varying portion of acid-secreting fundic glands might well be removed, along with the antrum mucosa. For this reason, as fourth and fifth stages in the experiment, the remaining gastric remnant was removed in part or completely. The first experiment with Animal D-628 (Fig. 9) provided suggestive evidence that removal of this fundic mucosa actually increased the output of gastric juice from the pouch in this preparation. In Animal D-628 the output of gastric juice increased somewhat after transplantation of the distal half of the antrum into the colon. However, when the remaining gastric remnant of antrum was excised, the output of gastric juice decreased significantly. When, then, one-half of the remaining remnant of stomach was excised, a significant increase in the output of gastric acid resulted. The data with Animal D-687 (Fig. 10) are difficult to interpret. After transplantation of the distal half of the antrum into the colon, a marked increase in the output of acid from the Heidenhain pouch occurred. When, however, the proximal portion of the antrum was removed from the stomach, the secretion of gastric juice from the Heidenhain pouch practically ceased and did not increase during subsequent stages of the experiment. At autopsy on this animal the Heidenhain pouch was found to be mark-

Fig. 11.—D-731



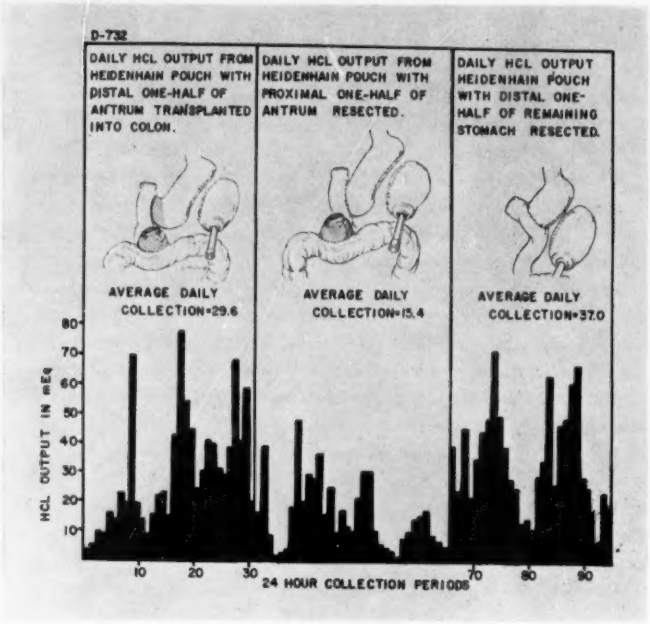
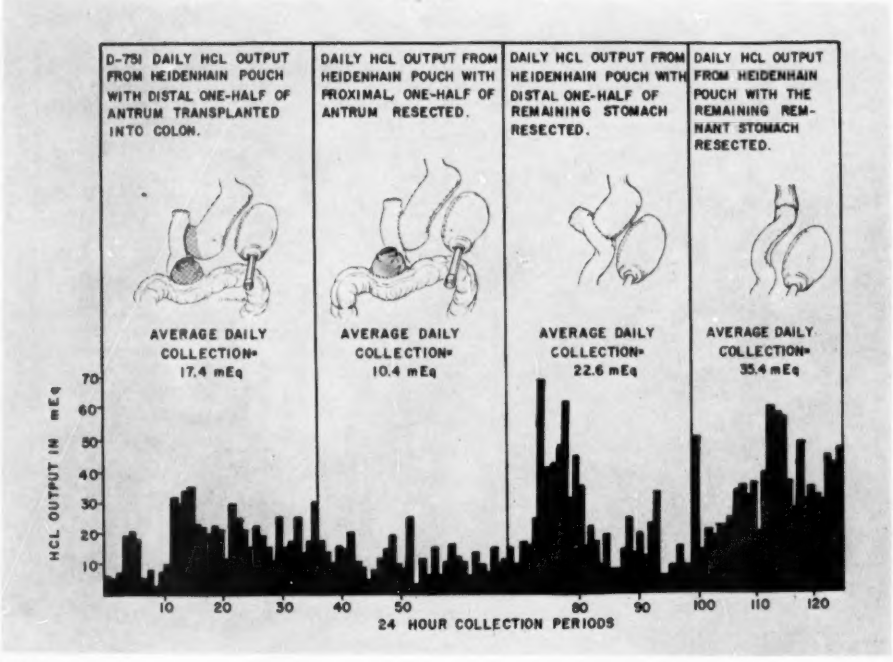


Fig. 12.—D-732

Fig. 13.—D-751



edly reduced in size, probably owing to impairment of its blood supply. The data secured in the remaining three animals, illustrated in Figures 11, 12, and 13, are all consistent and demonstrate that removal of acid-secreting fundic mucosa increases the output of acid from the Heidenhain pouch. In Animal D-731 (Fig. 11) a control measurement of the Heidenhain-pouch secretion when the entire antrum was in its normal location was not made. However, a fairly abundant secretion of gastric juice was present when the distal half of the antrum was transplanted into the colon. Following removal of the proximal portion of antrum from the stomach, the output of acid from the Heidenhain pouch increased significantly. However, as may be seen in the third column, when half of the remaining fundus of the stomach was excised, a more marked increase in gastric secretion from the Heidenhain pouch occurred. Complete removal of the gastric remnant was followed by a slight decrease in the output of gastric juice. The data secured with Animal D-732 (Fig. 12) are quite similar, except that in this animal there was no increase in gastric secretion when the proximal half of the antrum was removed from the stomach. However, when subsequent removal of one-half of the acid-secreting fundic remnant was done, the output of acid by the pouch increased markedly. In certain respects, the experiment with Animal D-751 (Fig. 13) is the most convincing. In this animal a satisfactory secretion of gastric juice by the Heidenhain pouch was induced when the distal half of the antrum was transplanted into the colon. This secretion fell when the proximal portion of the antrum was excised from the stomach. When half of the remaining fundus of the stomach was removed, the output of acid from the Heidenhain pouch increased markedly; and when the last remnant of stomach was removed, the output of acid was still further increased.

Comment

In 1953 Evans, Zubiran, McCarthy, Ragins, Woodward, and Dragstedt [12] found that partial or complete removal of the fundus of the stomach in Heidenhain-pouch dogs produced a profound increase in the secretion of gastric juice from the pouch. In this experiment, after removal of the fundus, the esophagus was anastomosed to the antrum of the stomach. The increase in secretion from the Heidenhain pouch was interpreted as due to the stimulating effect of neutral food in contact with the antrum and the absence of inhibition of this secretion which usually takes place when the food becomes acid in reaction. In the present experiments the antrum has been removed; so the increase in secretion from the Heidenhain pouch which follows removal of the acid-secreting fundus must be due to decreased inhibition of gastric secretion resulting from a decrease in the acidity of the food which comes in contact with the duodenum.

The experiment described by Harrison and his associates was repeated by us in 13 animals. In four of these, removal of the proximal portion of the antrum remaining in contact with the fundus was followed by an increase in the output of acid from the Heidenhain pouch. In the remaining nine dogs, however, no increase in secretion occurred, or there was an actual reduction. The experiment with Dog D-731, illustrated in Figure 11, is especially instructive. This animal put out an average of 19.5 mEq. of acid per day from the Heidenhain pouch when the distal half of the antrum was transplanted into the colon. This increased to 27.5 mEq. per day when the proximal portion of the antrum was removed from the stomach. However, when the distal one-half of the remaining acid-secreting fundus was resected, the average daily secretion increased further to 37 mEq. In all five of the animals, in which subsequent removal of all or portions of the remaining acid-secreting fundus was done, an increase in secretion from the Heidenhain pouch followed.

It must be admitted that the experiment of Harrison and his associates provided the best physiological evidence of an inhibitory

hormone from the gastric antrum. However, the negative results secured in nine dogs, with this type of experiment in our hands, and the alternative explanation afforded by the increase in secretion resulting from removal of the fundic mucosa make this evidence less impressive. Histological examination was made of the resected proximal portion of the antrum in each experiment. Portions of fundic mucosa in varying amounts were always found. Indeed, resection of the remaining antrum was purposely generous to make sure to include all of the antrum tissue. It may well be that a similar effort was made by Harrison and his associates. We believe that the increase in secretion, occasionally observed when the proximal antrum is excised in this experiment, is due not to removal of the antrum and its hypothetical inhibitory hormone but, rather, to removal of varying portions of the acid-secreting fundus, which results in decreased inhibition of gastric secretion from the duodenum. Such an explanation is in harmony with our failure to demonstrate an inhibitory hormone in the experiments in dogs with a Heidenhain pouch and two isolated pouches of the antrum. It is also in harmony with the finding that the introduction of acid food into the isolated antrum pouch blocks the secretion of gastric juice from the Heidenhain pouch immediately, rather than after a preliminary period of stimulation. Such a preliminary period of stimulation might be expected from the presence of food in the antrum and a secondary inhibition of secretion at a later period, due to the release of an antrum inhibitory hormone. The effect of acid in contact with the antrum mucosa more nearly resembles the effect of the application of cocaine. The simpler explanation would be that both acid and cocaine block the formation or release of gastrin.

It must be emphasized that in all of these experiments the target organ, namely, the Heidenhain pouch, is devoid of vagus innervation. It is quite conceivable that inhibitory effects on vagus-induced gastric secretion might occur.

Summary and Conclusions

The experiment described by Harrison, Lakey, and Hyde, which was interpreted to demonstrate an inhibitory hormone from the gastric antrum, was repeated in 13 dogs. In four of these, with the distal half of the antrum transplanted into the colon as a diverticulum, removal of the proximal antrum, which had been left in continuity with the fundus, increased the output of acid from the Heidenhain pouch, whereas in nine dogs the acid output remained the same or was decreased. However, further removal of fundic mucosa in five of these animals produced an augmentation in secretion from the Heidenhain pouch. It is concluded that the increase in secretion, observed in some of the animals when the proximal half of the antrum was excised, is due to removal of varying portions of the acid-secreting fundus rather than to excision of the antrum tissue itself. The findings in the present experiment support the view, previously expressed, that acid in contact with the mucosa of the gastric antrum blocks the formation or release of gastrin in a manner perhaps similar to that of cocaine. An inhibitory hormone from the gastric antrum is not demonstrated by this type of experiment.

Department of Surgery, The University of Chicago, 950 E. 59th St. (37).

REFERENCES

1. Babkin, B. P.: Secretory Mechanisms of the Digestive Glands, Ed. 2, New York, Paul B. Hoeber, Inc. (Medical Book Department of Harper & Brothers), 1950, p. 642.

2. Wilhelmj, C. M.; McCarthy, H. H., and Hill, F. C.: Acid Inhibition of the Intestinal and Intragastric Chemical Phases of Gastric Secretion, Am. J. Physiol. 118:766, 1937.

3. Wilhelmj, C. M.; O'Brien, F. T., and Hill, F. C.: Influence of the Pylorus on the Secretion of Acid by the Fundus, Am. J. Physiol. 116:685, 1936.

4. Wilhelmj, C. M.; Finegan, R. W., and Hill, F. C.: The Physiological Control of Gastric Acidity, Am. J. Digest. Dis. & Nutrition 4:547, 1937.

5. Oberhelman, H. A., Jr.; Woodward, E. R.; Zubiran, J. M., and Dragstedt, L. R.: Physiology of the Gastric Antrum, Am. J. Physiol. 169:738, 1952.

6. Woodward, E. R.; Lyon, E. S.; Landor, J., and Dragstedt, L. R.: Experimental Studies on Isolated Antrum Pouches in Dogs, Gastroenterology 27:766, 1954.

7. Brunschwig, A.; Rasmussen, R. A.; Camp, E. J., and Moe, R.: Gastric Secretory Depressant in Gastric Juice, Surgery 12:887, 1942.

8. Hood, R. T., Jr.; Code, C. F., and Grindlay, J. H.: Source of a Possible Gastric Secretory Inhibitor in Canine Gastric Juice and Effects of Vagotomy on Its Production, Am. J. Physiol. 173:270, 1953.

9. Harrison, R. C.; Lakey, W. H., and Hyde, H. A.: Production of an Acid Inhibitor by the Gastric Antrum, Ann. Surg. 144:441, 1956.

10. Dragstedt, L. R.; Woodward, E. R.; Storer, E. H.; Oberhelman, H. A., Jr., and Smith, C. A.: Quantitative Studies on the Mechanism of Gastric Secretion in Health and Disease, Ann. Surg. 132: 626, 1950.

11. Longhi, E. H.; Greenlee, H. B.; Bravo, J. L.; Guerrero, J. D., and Dragstedt, L. R.: Question of an Inhibitory Hormone from the Gastric Antrum, Am. J. Physiol. 191:64, 1957.

12. Evans, S. O., Jr.; Zubiran, J. M.; McCarthy, J. D.; Ragins, H.; Woodward, E. R., and Dragstedt, L. R.: Stimulating Effect of Vagotomy on Gastric Secretion in Heidenhain Pouch Dogs, Am. J. Physiol. 174:219, 1953.

Small-Intestinal Gallstone Ileus and Use of Barium Sulfate per Os in Its Diagnosis

VICTOR DRUCKER, M.D., Binghamton, N. Y.

An unequivocal preoperative diagnosis of small-intestinal ileus is very often difficult to make, and a positive determination of the etiology of such obstruction is usually impossible. Once a fairly certain diagnosis has been established, it becomes extremely important to discover the source of the trouble without unnecessary delay, for the correct management of the condition may depend upon the identification of the causative agent. Certain mechanical small-bowel obstructions can be treated conservatively, deliberately, and nonsurgically, with complete symptomatic relief, by decompression suction therapy. In most cases of small-intestinal ileus, however, dilatory procedures are contraindicated and procrastination is hazardous.

The delay is particularly dangerous when such ileus is caused by an impacted gallstone, for the mortality rate in cholelithic obstruction has been reported as varying from 30% to 70%, owing, in great part, to the tardiness in diagnosis and treatment.[5] Since it is an accepted fact that radiologic examination of the abdomen is the best presently used method of making an accurate preoperative diagnosis of gallstone ileus, it is reasonable to assume that a more searching x-ray investigation might expedite the diagnosis and appreciably lower that distressing mortality figure.

The criteria for the roentgen diagnosis of gallstone ileus from the flat-plate studies of the abdomen are as follows:

(*a*) Distended loops of small intestine—the basic roentgen sign of small-bowell obstruction

(*b*) Air in the biliary tract (gallbladder, ducts, and radicles)—evidence of a fistula

(*c*) Absence of gallstone previously seen within a gallbladder that had not been removed in the interim—suggestive of migration of the stone following its passage from the viscus

(*d*) Presence of gallstone in an unusual location—indicative of an earlier perforation with movement of the stone

(*e*) Visualization of impacted calculus against a background of gas in the obstructed bowel

Yet, with all of these helpful radiographic features, it is somewhat surprising that the films so frequently are nondiagnostic. This may be due to any one of several reasons.

1. At the outset, gallstone ileus is intermittent, arising when the stone halts and subsiding when it moves along. If the radiograph is taken during an episode of movement, there may be very little or no small-bowel distention.

2. The biliary fistula may close over before the obstruction occurs, and gas will not be found in the biliary tract.

3. The majority of gallstones are radiolucent and are, therefore, undetectable on the flat-plate studies of the abdomen.

4. The fluid- and gas-filled bowel loops may so overlap that the calculus, radiolucent or radiopaque, is completely obscured.

It is evident, therefore, that many or all of the roentgen aids and guides detailed above may be lacking and no information be available. However, if the scout films of the abdomen are supplemented by x-ray studies taken after the proper use of radiopaque contrast material, the chances of directly, or indirectly, demonstrating the stone will be considerably increased. The calculus, even though radiolucent, may appear in the blocked bowel either as a filling defect in a surrounding zone of opacification—just as it might be disclosed within the functioning gallbladder on cholecystography

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From the Department of Radiology, Binghamton City Hospital.

—or as an intraluminal body that has been invested with a thin coat of opacity.

On reviewing the roentgenograms in two cases of preoperatively diagnosed gallstone ileus, in which barium had been given earlier, I was impressed by the fact that it was the opaque medium profiling the borders of the calculi that had made the diagnoses simple and certain (Figs. 1, 2, 3, and 4). This is graphically brought out in Figure 4 (Case 2). The stone could never have been otherwise demonstrated within the intestine, for a radiograph of the removed and washed large specimen shows it to be practically radiolucent (Fig. 5).

The thought occurred that if a contrast agent were to be given at the outset, with the primary intention of delineating the impacted gallstone, a positive diagnosis of small-bowel cholelithic obstruction might be made earlier and definitive surgery instituted more promptly.

Which one of the various radiopaque media should be used? How could it be best administered?

Among the groups of contrast substances that might be employed are thorium dioxide compounds (Thorotrast [a stabilized colloidal suspension of thorium dioxide], Umbrathor [a 25% suspension of thorium oxide]), which remain fluid[2]; iodized oils (chloriodized oil [Iodochlorol], iodized oil [Lipiodol]); iodinated water-soluble dyes (acetrizoate [Urokon], diatrizoate [Hypaque], Renografin*), such as are now used in urographic studies,[3,4,6,7,10,15,20] and barium sulfate suspensions.[1,8,9,16]

The ideal material should be easy to take and show good detail on the roentgenogram; it must be innocuous in the intestinal tract or peritoneal cavity in case of perforation or spill, and must not give rise to any new condition inimical to the contemplated surgery. Although the agents mentioned satisfy most of these requirements, all have a few undesirable features—qualities which would limit, or even interdict, their use in many instances.

Thorium dioxide and its derivatives possess good radiographic contrast but have the very serious disadvantages of being retained indefinitely within the reticuloendothelial system and of being radioactive, thereby giving rise to a definite radiation hazard. Long after its instillation or injection in retrograde pyelography, salpingography, bronchography, or arteriography, the material has been found in the regional tissues, lymph nodes, liver, and spleen as a granuloma or as retained particles. Fibrosis, necrosis, abnormal calcifications, and hematopoietic disturbances have been reported as sequelae. Radioactivity was proved by gamma-ray counting and radioautography of the spleen.[19,22]

Iodized oils are densely radiopaque, but are unpleasant to the taste, and are prone to separate into globular collections so as to rather indistinctly delineate the mucosal lining.

Urographic media, slightly modified for use as oral gastrointestinal contrast agents, have been found to have fairly good density, and to be relatively nontoxic; they are not readily absorbed, and, because of their low viscosity, may demonstrate small fistulous tracts. At the same time, they have been reported to increase peristalsis and create a rapid transit time, to be somewhat irritating to the intestine and to cause diarrhea in the nonobstructed or partially obstructed bowel, to give poor definition to the mucosal pattern in the ileum and most of the jejunum, to have a bitter taste, and, finally, to be relatively expensive in the amounts required.

Barium sulfate, one of the first of the radiopaque media to have been adapted for use in the gastrointestinal tract, is very easy to administer and presents excellent radiographic detail; it is nonabsorbable and nontoxic, does not have an actually disagreeable taste, and is inexpensive. However, it may fail to fill or to outline small fistulous sinuses; it is not water-soluble, tends to precipitate out, and may bring about a barium impaction in the colon subsequent

* Renografin is a mixture of sodium and methylglucamine salts of diatrizoate.

to absorption of its aqueous vehicle in the large bowel. Unfortunately, detractive qualities that have been erroneously ascribed to it in the past have needlessly restricted its use. The older literature is punctuated with statements to the effect that barium should never be given by mouth in cases of suspected obstruction.[18,23] For a long time surgeons and radiologists were of the opinion that the barium suspension would plug the bowel in the region of the lesion and transform a partial intestinal obstruction into a complete one.[1,3] Such conversions, occurring in the large bowel,[21] are understandable, for the colon, through its basic physiological property of absorption, removes the water from the barium suspension, leaving a particulate precipitate, which might well give rise to an impaction there.[17] The small intestine, on the other hand, not only is devoid of hygroscopic powers but forms additional fluid in irritative response to the obstructive process; thus, the barium sulfate suspension becomes even more dilute, and the chances for a barium-created complete small-bowel obstruction are rather remote. In support of this contention are the numerous reported cases in which barium sulfate was administered orally by mistake without any ill effects. Indeed, more recently, many radiologists [1,5,6,8,16,24] have talked about the intentional, not the inadvertent, use of barium sulfate suspensions in suspected intestinal obstruction, with a qualifying recommendation that the colon first be excluded as the site of the obstruction.

Another earlier objection to the use of barium was that if perforation of the stomach or intestine occurred during a barium study, peritonitis resulted, which was due to the chemical in the suspension.[10,20] Kleinsasser and Warshaw [14] had perceived that when perforation occurred in the stomach or duodenum in the course of a barium-meal examination, the patient made an uneventful recovery following operation for closure of the ruptured viscus, but when the colon was perforated, the ensuing peritonitis was almost invariably fatal. Postulating that death was due to a bacterial contamination from the large bowel rather than to a chemical irritation of the peritoneum, they performed a series of interesting experiments in which dogs were given intraperitoneal injections of heavy barium sulfate solutions, mixtures of barium sulfate and stool, ordinary stool, and sterile stool. None of the animals receiving the barium sulfate or sterile stool preparations died, but the dogs which had injections of untreated stool promptly died. Operations on the animals that lived disclosed barium sulfate granulomata on the surfaces of the intestine and peritoneum, as well as diffuse inflammatory changes in the peritoneum. Kay [13] reported that barium sulfate particles accidentally spilled into the peritoneal cavity of humans gave rise to slight inflammatory and fibrous tissue reactions. He felt that a fibrous tissue granuloma which had developed in one of his cases would probably have eventually disappeared spontaneously. Huston [12] found no sign of fibrosis or other pulmonary-tissue abnormality four months after the experimental introduction of barium sulfate into the lungs of rats. Observations such as these warrant a conclusion that barium within the body cavities, particularly in the peritoneal space, can be safely left alone and should cause no concern.[11]

Because all of the afore-mentioned media are far from being gastronomic delights and because the patient, a priori, is nauseated and vomiting, the administration and retention of the diagnostic agent pose quite a problem. Even if the patient can ingest and retain a reasonable quantity, the medium may be spread so thin through a markedly distended small bowel that the amount collected and concentrated at the site of the obstruction will be insufficient to reveal the cause of the trouble. To obviate these difficulties, the material may be instilled through a long intestinal tube that has previously been passed down to the point of obstruction or close to it.

In addition to functioning as a carrier for the diagnostic agent, the tube serves to

decompress the dilated small intestine and thereby affords a modicum of relief to the stricken patient. Should the surgeon wish to have the blocked segment completely clean of medium at operation, or if he is still somewhat skeptical anent the harmlessness of barium remaining within the obstructed small bowel, the medium may be removed via the same tube by warm-water irrigation and aspiration, as recommended by Cantor.[1]

Assuming that the issues of suitable contrast agent and practical method of administration have been resolved, I suggest the following procedure:

A long intestinal tube (Cantor or Miller-Abbott) is passed well down into the small bowel, and its progress and position are checked by roentgenographic examination. If there is appreciable small-bowel distention, suction decompression is effected; it is not necessary to obtain complete decompression or to try to get the tip of the tube to the precise point of obstruction. Barring any marked delay in the stomach, the tube should be well within the small intestine in about 12 hours, or slightly more. When this position has been established roentgenographically, 125 to 150 cc. of dilute barium sulfate suspension is instilled through the tube and further suction is discontinued. Films are taken one and three hours later; if necessary, additional roentgenograms are obtained at about three- to four-hour intervals until the medium has either completely stopped at the area of occlusion in the small bowel or passed on to enter the colon. The barium is allowed to remain at the site of obstruction for two or three hours—long enough for it to outline the contours of the agent of obstruction. After that time, the barium sulfate can be removed by gentle warm-water irrigation and aspiration through the tube.

The proceedings described above may seem to add to the presently unavoidable, and almost deplorable, delay before surgical intervention, but they actually save considerable time by substantially shortening the interval between the patient's hospital admission and the establishment of a definite diagnosis. This will hold true in almost all cases, although those of us who have had any experience with intestinal intubation know that, on occasions, it may be a matter of a couple of days before the intestinal tube gets down to the region of the distal ileum. This "hold-up," when it occurs, has no serious implications, but it can be extremely exasperating—to the patient even more than to the physician. The protracted intubation not only may irritate the patient's nose and throat, but may also affect his frame of mind, disturbing his equanimity and tolerance to such a degree that it may be well-nigh impossible to restrain him from tearing out the tube as soon as he is left alone.

I should like to restate that aspiration of the obstructed-bowel contents must be suspended as soon as the barium solution has been admitted through the tube, because suction drainage after that time will remove the medium and completely nullify the diagnostic value of the procedure. To repeat so self-evident a fact may seem unnecessary, and almost tautological; yet I believe that the emphasis is justifiable, first, because with seriously ill patients—as these usually are—therapy tends to be continous and nursing care uninterrupted, and, second, because just such an instance of misdirected activity actually occurred in a recent case of gallstone ileus that was being studied by the above intubation method. (A belated inquiry

Fig. 1 (Case 1; cited but not reported here).—Distended small bowel filled with dilute barium sulfate suspension. Round mass faintly shown in pelvis (delimited by arrows) was originally interpreted as either a gallstone or a partially calcified fibroid of the uterus.

and postoperative review of the charted nurse's notes revealed the error in technique.)

One of the two cases of small-bowel obstruction in which barium sulfate suspension given early had made the preoperative diagnosis of gallstone ileus very easy was the basis for a previous article. Two films from that case (Figs. 1 and 2) demonstrate clearly how the medium on the surface of the calculus helps in identification of the stone. The second, a more recent, case has some unusual features and particularly representative radiographs, so that it may be worth while to report it here.

Report of a Case

A 72-year-old white man was admitted to Binghamton City Hospital on July 10, 1957, with the complaint of upper abdominal pain, nausea, and vomiting of blood.

The past history disclosed that for more than 10 years he had experienced epigastric distress and heartburn, which were relieved by alkalis, and upper abdominal soreness after eating fatty foods. There had been some constipation, but no anorexia or loss of weight. Twenty years prior to admission he had had an operation for glaucoma, and, more recently, an operation for an enlarged benign prostate.

His present illness began on the night before admission with slight nausea and nonradiating epigastric pain, followed early next morning by a short bout of hematemesis. Because he was blind, he could not tell whether or not his stools had been tarry or had contained bright blood. That there had been hematemesis was suggested by his statement that the "vomit tasted bloody"; the occurrence of hemorrhage was confirmed by his landlady, who had seen the vomitus.

Physical examination showed the patient to be well nourished, not in acute distress, and not icteric. Save for blindness, the head was normal; the heart and lungs were negative. Blood pressure was 140/90; pulse 96 per minute and regular; temperature 37.3 C (99.8 F). The abdomen presented a healed suprapubic scar and was not distended. Slight, nonrebound tenderness was elicited over the epigastrium, but no abnormal masses were palpable. Laboratory examinations were reported as follows: hemoglobin 97%; hematocrit 45%; white cell count 15,000 per cubic millimeter; trace of acetone and 2 to 3 white and red blood cells in the urine; NPN 53 mg. %; sugar 155 mg. %; sulfobromophthalein (Bromsulphalein) retention increased to 33% at 45 minutes. A limited emergency gastrointestinal roentgen-ray examination showed pylorospasm, spasticity of the duodenal bulb, and a suggestive ulcer-crater defect near the

Fig. 2 (Case 1).—Tip of Miller-Abbott tube in distal ileum. Small-bowel distention considerably decreased; contrast material in terminal ileum and ascending colon. Pelvic mass, lined by medium, is now unmistakably seen to be a biliary calculus.

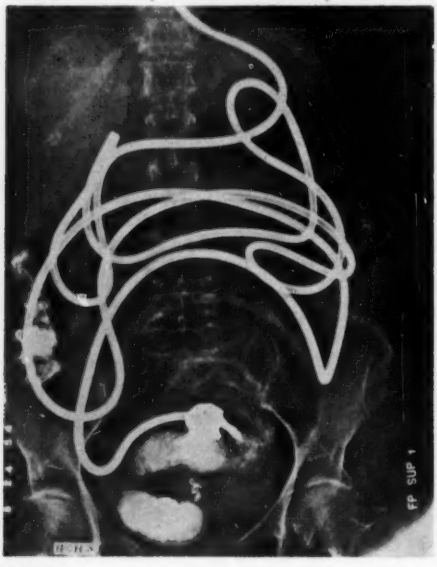
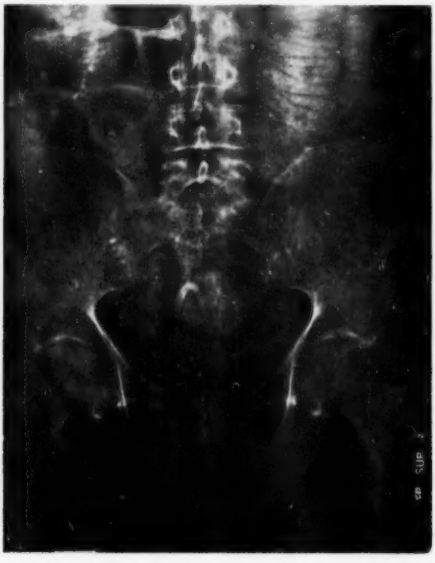


Fig. 3 (Case 2, reported herewith).—Barium sulfate suspension in dilated small intestine. Large, round biliary calculus partially covered by the medium in right side of pelvic cavity. Catheter in urinary bladder.



base of the bulb on the margin of its lesser curvature.

During the first two days of hospitalization, he vomited several times, but there was no hematemesis. Levin-tube drainage on July 12 did not yield any blood, and a soapsuds enema evacuated partly formed brown stool, also without evidence of blood. A flat plate of the abdomen disclosed moderately dilated small-bowel loops and barium (from the gastrointestinal series of July 10) in the proximal colon, indicating that the small-intestinal obstruction was not complete. On July 14, cramplike pain in the right upper quadrant, with rebound tenderness and abdominal distention, set in. A roentgenogram at this time (Fig. 3) revealed increased small-bowel dilatation and a large, ovoid, radiolucent mass with a radiopaque periphery, which was interpreted as a gallstone which had become impacted and caused the ileus. On July 15, the Levin tube was removed and a Miller-Abbott tube was passed, with minimal relief. A series of radiographs from July 15 to July 17 showed slow progression of the Miller-Abbott tube, slight onward movement of the calculus, and gradually increasing dilatation of the small intestine.

With the diagnosis established and the patient properly prepared, laparotomy was performed on July 17. The small bowel was found to be markedly distended, and the point of obstruction was identified about 40 cm. proximal to the ileocecal valve. The bowel at the site of the occlusion was exteriorized, and two gallstones were removed by enterotomy. One stone was large and ovoid and measured 3.5×3×3 cm.; the other was small, round, and 1.3 cm. in diameter. The entire small bowel and colon were palpated, but no other calculi were found. The gallbladder could not be well examined because of numerous adhesions in the area. The abdomen was closed and the patient sent back to the floor.

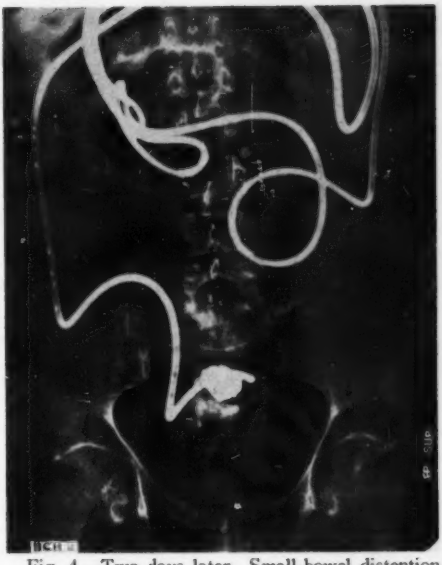


Fig. 4.—Two days later. Small-bowel distention is diminished; only a trace of barium remains, diffusely dispersed through the distal bowel. Calculi, circumscribed by barium sulfate, are strikingly demonstrated.

Fig. 5.—Calculi of reported case removed at operation. Both are shown to be almost completely radiolucent.

Three weeks after operation, oral and intravenous radiographic examinations of the biliary tract failed to show a gallbladder shadow, but the common duct was outlined and seen to be normal. A gastrointestinal series a few days later disclosed a spastic and irritable duodenal cap; there was no evidence of a cholecystoduodenal fistula.

Convalescence was uneventful, and the patient was discharged on Aug. 15, in good condition.

This case is somewhat unusual because the chief presenting sign of hematemesis is uncommon in cholecystic disease. It is, of course, possible that the patient had a concomitant active peptic ulcer of the duodenal bulb. It is more logical, however, to assume that the bleeding was caused by the calculus breaching the duodenum, albeit only a very small percentage of gastrointestinal hemorrhages is due to cholelithiasis.

Since the patient, on admission, did not show any signs of continuing hemorrhage and his blood picture was fairly good, the radiologist suggested that scout films of the abdomen be first taken and that a routine gastrointestinal series be done in a day or so, at which time the patient would be better prepared for a thorough and unhurried study. Had this recommendation been heeded, an exemplary serial study of gallstone ileus might have been obtained. However, an emergency roentgenographic examination of the upper gastrointestinal tract was urgently requested late on the afternoon of admission. The examination was, perforce, limited and somewhat unsatisfactory; but there was no indication of a gallstone on any of the films, and the available evidence pointed to a diagnosis of active duodenal peptic ulcer. The next requested radiograph of the abdomen, taken three days later, showed that much of the barium had moved along to enter the colon. The roentgenograms in the case are interesting and support the argument that barium sulfate suspension does not settle at the obstructing lesion, to change a partial small-intestinal ileus into a complete one. Even more pertinent to the subject is the vivid portrayal of contrast medium coating the gallstones (Fig. 4) and proving a major premise in our presentation.

Comment

Mechanical small-bowel obstruction, of whatever etiology, is hard to diagnose, and is usually very serious because of the long period intervening between the time of the patient's arrival at the hospital and the physician's arrival at the diagnosis. In cases where such obstruction has been precipitated by an impacted gallstone, the mortality figures are extremely high—this, in spite of the fact that there are several radiographic signs which, when present, could make the diagnosis of cholelithic ileus fairly simple. These clues, unfortunately, are not invariably evident on the routine scout film of the abdomen. However, if contrast material

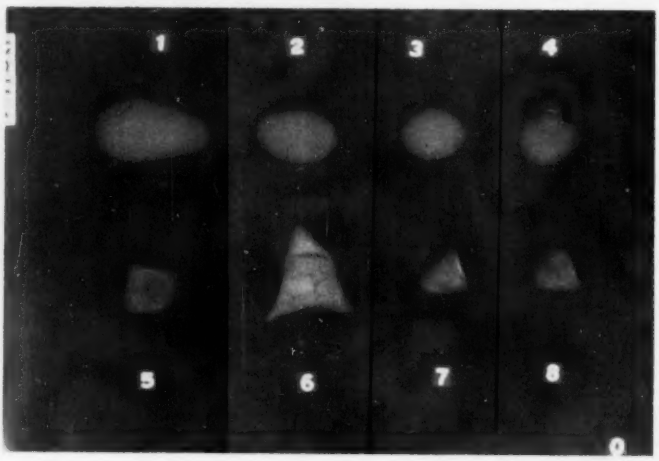


Fig. 6.—Collection of eight different radiolucent and slightly calcified gallstones. Stones 1 to 4 possess rough surfaces; stones 5 to 8 have smooth faces.

is introduced into the involved portion of obstructed intestine, the offending calculus may be sharply limned on the roentgenogram. This is strikingly demonstrated on one of the radiographs (Fig. 4) of the case reported above.

To verify the intensifying effect of an opaque coat on an inconspicuous calculus, I performed a rather simple experiment. Radiographs of a large number of various types of gallstones that had been stored in our pathology department were taken and eight radiolucent or very faintly calcified stones selected (Fig. 6). Four had rounded borders and rough superficies; the other four were faceted and had smooth exteriors. Their dimensions and diameters ranged from 1.5 to 4 cm. Sets of two stones, one coarse-surfaced and one even-faced, were immersed for four hours in different radiopaque substances, then removed and again x-rayed in almost the identical positions (Fig. 7). Even casual comparison of the "dry" and "wet" specimens shows how the contrast agents circumscribe and silhouette the stones.

Of the many new and old radiopaque preparations, the barium sulfate suspension would seem to be the most satisfactory. Without extolling its radiographic attributes, barium should be given precedence over the other, more modish media, because it has a minimum of shortcomings and a maximum of desirable qualifications.

An early major objection to the use of barium sulfate was the fear that if the material accidentally entered the body cavities, a grave infection would result. This fear has not been borne out clinically or experimentally. It is now understood that the danger is created by the consequent bacterial contamination, and not by the immediate spill per se. Because barium sulfate is water-insoluble and disposed to settle, it has been unjustly impugned and incorrectly accused of transforming a partial intestinal obstruction into a complete one; but such a change has not been found to occur in the small bowel. It is, in fact, by virtue of its physicochemical properties, that barium sulfate suspension is so well suited to serve as an aid in the diagnosis of gallstone ileus, for the process of sedimentation may deposit fine barium sulfate particles on the usually uneven surfaces of the obstructing stone so as better to delineate and demonstrate it on the roentgenogram.

The diagnostic and therapeutic use of a long intestinal tube in small-bowel obstruction has been described by numerous authors.[1,16,17] Osgood[17] declares that the principal value of a barium injection through the tube lies in disclosing the presence of an obstruction rather than its cause, although he states that a sharp angulation at the point of occlusion suggests adhesions there. It is our opinion, however, that other elements re-

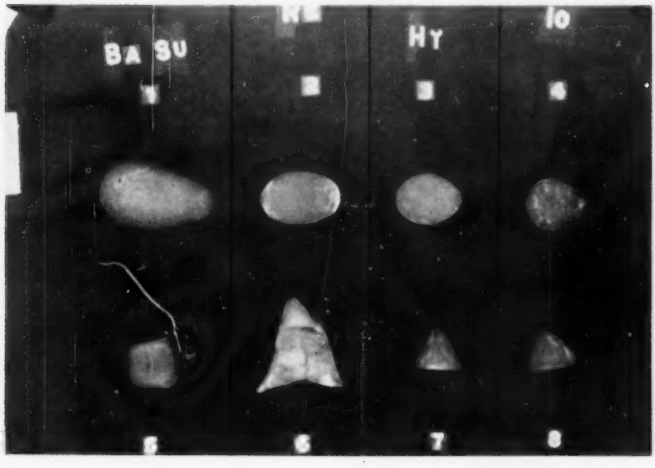


Fig. 7. — Same gallstones after immersion for four hours in dilute barium sulfate suspension —Renografin 60%, diatrizoate (Hypaque) sodium 50%, and chloriodized oil (Iodochlorol). The radiopaque materials profile the calculi and aid in their detection.

sponsible for small-intestinal ileus can also be identified by this method, particularly when the precipitating agent is an impacted gallstone.

Summary

The types, etiology, and radiographic appearance of small-intestinal obstruction are briefly outlined. Preoperative diagnosis is not easy, but may be facilitated by the peroral use of radiographic contrast media; this applies especially to cases of gallstone ileus, wherein the occluding stone is rimmed and ringed by the opaque material.

The advantages and disadvantages of various radiopaque agents are enumerated, of which barium sulfate suspension has been found to be the most satisfactory. The early fears of possible complications arising from its use are shown to be groundless.

A simple and effective diagnostic procedure which utilizes intestinal intubation and instillation of dilute barium sulfate suspension through the tube is described.

A case of preoperatively diagnosed gallstone ileus with representative radiographs is presented.

Figures 1 to 4, inclusive, are reproduced by means of the LogEtronic Printer.

Binghamton City Hospital.

REFERENCES

1. Cantor, M. O.; McCollum, B. E., and Hodges, J.: Intestinal Intubation for Barium Produced Bowel Obstruction, Am. J. Digest. Dis. 19:148, 1952.

2. Case, J. T.: Umbrathor as a Substitute for Barium in the Roentgen Study of Acute Intestinal Obstruction, Am. J. Roentgenol. 58:422, 1947.

3. Davis, L. A.; Huang, K-C., and Pirkey, E. L.: Water-Soluble, Nonabsorbable Radiopaque Mediums in Gastrointestinal Examination, J. A. M. A. 160:373, 1956.

4. Davis, L. A.: Use of Soluble Non-Absorbable Opaque Media in the Examination of the Gastrointestinal Tract in Children, South. M. J. 49:1011, 1956.

5. Drucker, V., and McElwee, C. H.: Recurrent Cholelithic Intestinal Obstruction, Gastroenterology 32:927, 1957.

6. Epstein, B. S.: Nonabsorbable, Water-Soluble Contrast Mediums: Their Use in Diagnosis of Intestinal Obstruction, J. A. M. A. 165:44, 1957.

7. Epstein, B. S.: The Use of a Water Soluble Contrast Medium (Hypaque) for Gastrointestinal Roentgenography, Am. J. Roentgenol. 78:694, 1957.

8. Frimann-Dahl, J.: Administration of Barium Orally in Acute Obstruction: Advantages and Risks, Acta radiol. 42:285, 1954.

9. Fulton, H.: The Gastrointestinal Radiologist, Editorial, Am. J. Roentgenol. 77:745, 1957.

10. Gipson, E. N.: Preliminary Report on the Use of Oral Renografin (Gastrografin) in Gastrointestinal Studies, Monographs on Therapy, Squibb Institute for Medical Research, New Brunswick, N. J., 1957, p. 242.

11. Hayden, R. S.: Perforation of Duodenal Ulcer During Fluoroscopy, Radiology 57:214, 1951.

12. Huston, J., Jr.; Wallach, D. P., and Cunningham, G. J.: Pulmonary Reaction to Barium Sulfate in Rats, A. M. A. Arch. Path. 54:430, 1952.

13. Kay, S.: Tissue Reaction to Barium Sulfate Contrast Medium—Histopathologic Study, A. M. A. Arch. Path. 57:279, 1954.

14. Kleinsasser, L. J., and Warshaw, H.: Perforation of the Sigmoid Colon During Barium Enema, Ann. Surg. 135:560, 1952.

15. Love, L.: Oral Use of Renografin, Monographs on Therapy, Squibb Institute for Medical Research, New Brunswick, N. J., 1957, p. 260.

16. Martin, J. F., and Friedell, H. L.: Partial Obstruction of the Small Intestine, Radiology 59: 49, 1952.

17. Osgood, E. C.: The Role of the Radiologist in the Management of Patients with Intestinal Obstruction, with Special Reference to the Use of the Miller-Abbott Tube, Radiology 49:529, 1947.

18. Rigler, L. G.; Borman, C. N., and Noble, J. F.: Gallstone Obstruction: Pathogenesis and Roentgen Manifestations, J. A. M. A. 117:1753, 1941.

19. Roberts, J. C., Jr., and Carlson, K. E.: Hepatic Duct Carcinoma 17 Years After Injection of Thorium Dioxide, A. M. A. Arch. Path. 62:1, 1956.

20. Shehadi, W. H.: Use of Oral Renografin (Gastrografin) in the Examination of the Gastrointestinal Tract, Monographs on Therapy, Squibb Institute for Medical Research, New Brunswick, N. J., 1957, p. 233.

21. Stalker, L. K.: Barium as a Factor in Intestinal Obstruction, Am. J. Surg. 72:756, 1946.

22. Vögtlin, J., and Minder, W.: Damage Due to Thorotrast After Bronchography, Retrograde Pyelography, Salpingography and Arteriography, Radiol. Clin., Basel, 21:96, 1952.

23. Weber, H. M., and Kirklin, B. R.: The Roentgenologic Investigation of the Small Intestine, M. Clin. North America 22:1059, 1938.

24. Zatzkin, H. R.; Tugendhaft, R. I., and Curran, H. P.: Roentgen Diagnosis of Spontaneous Internal Biliary Fistulas and Gallstone Ileus, Surg. Gynec. & Obst. 102:234, 1956.

Closed-Chest Resuscitation

ROBERT M. HOSLER, M.D., and KENNETH WOLFE, Cleveland

The most serious obstacle to overcome in present-day cardiorespiratory resuscitation is the necessity of opening the chest. This step of the procedure has always been a stumbling block, and no doubt has been one of the factors which retarded the development of resuscitation from 1902 to 1950. Only within the past six years has cardiac resuscitation become an acceptable procedure in the operating room. Finally, resuscitation emerged from its underground tunnel of despair to be accepted as a therapeutic measure with potential new horizons for overcoming temporary death and restoring life. Experiments now indicate that closed-chest resuscitation can become a practical reality.

Experience leads us to believe that the time has arrived when cardiac resuscitation must be included in certain cases of sudden clinical death (relative death [10]) outside the operating room, and possibly outside the hospital.

For example, resuscitation is now feasible for those persons facing impending death from acute coronary insufficiency [1-5] if the victim "dies" in the hospital. Previous preparation for such a procedure should be made before clinical death occurs. This should consist of having a mobile emergency resuscitation cart outside the room and a precise mental rehearsal by the resuscitation team. Otherwise, a frantic, inefficient atmosphere may prevail during the exigency. Grouping of these "coronary patients" in convenient areas of the hospital, instead of having them scattered around the hospital, is suggested. It would seem advisable to have a sterile preparation of one arm, swathing it then in alcoholic dressings in readiness for an intra-arterial perfusion.

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From the Claude S. Beck Laboratory for Cardiovascular Research.

It is the medical consensus that the great majority of patients who die suddenly of acute coronary insufficiency undergo terminal ventricular fibrillation. According to Yater and Beck, many of these hearts show minimal or no myocardial damage and may under proper circumstances beat again if given a second chance. Ideally, clinical death must overtake the victim where personnel with "resuscitative know-how" and proper equipment are in readiness. The death factor in the stage of clinical death may represent temporary death (relative death [10]), and as such may be slight and reversible. It may be likened at times to turning off the ignition of an engine, which, once it is restarted, may continue to function for an indefinite period.

Accomplishment of external defibrillation has been done for many years. However, it has not proved practical in restoring a circulation much beyond 45-55 seconds. Failure is not from inability to place the heart in defibrillation but from inability to restore a blood pressure compatible with life. Electrocardiographic evidence is readily obtained which indicates that the heart has been in defibrillation up to five or seven minutes after onset of the procedure. No pulse or blood pressure is recordable, and upon opening the chest the heart is found in standstill. In the meantime the heart is dying, although acceptable complexes are recorded upon the monitor.[6,7]

It naturally follows that what is needed at this juncture of the resuscitative procedure is a method of initiating effective spontaneous beats in this organ, preferably by not resorting to direct cardiac massage. Fifty years ago Crile,[8] of Cleveland, recommended and carried out brachial-artery perfusion with saline and epinephrine in order to combat cardiac standstill in the laboratory and the operating room.

TABLE 1.—*Electrodes Applied Directly to Myocardium*

No. of Dogs Used	Duration of Fibrillation, Sec.	EKG Defibrillation After Massage	Ventilation and Massage	Return of Circulation	Survivals
86	120	86	86	86	86

Experimental Study

The following animal experiments have been carried out to demonstrate various methods of resuscitation, their time limitations, and their practicalities. The last method (Table 4) would seem feasible, as it no doubt can be adapted to the human and may become the procedure of choice, particularly in medical and emergency wards.

In a series of 86 lightly anesthetized dogs (Table 1) the heart was placed in ventricular fibrillation by the direct application to its surface of a weak, 110-volt alternating current (about 25 ma.). In each instance manual massage and ventilation of the lungs was instituted exactly two minutes after the development of ventricular fibrillation. In each instance the normal heart rhythm was readily restored by an electrical countershock of 110 volts and approximately 2.5 amp. All 86 dogs survived for at least 72 hours and were normal in all respects within 24 hours.

External defibrillation of the heart with the intact chest was carried out in another series, of 55 dogs. The stoppage of the heart was put into effect by an electrical current of 110-220 volts supplied to the shaven chest of the animal. This cessation of the cardiorespiratory system was carried out under what was thought to be ideal circumstances for resuscitative procedures. The circulatory system was normal. Stoppage was sudden. Anesthesia had been light. Special instruments and oxygen equipment were at hand. No previous tests had been made upon the animals.

Ventricular fibrillation was determined by electrocardiographic records and confirmed by an abrupt fall of blood pressure to zero. The method of defibrillation consisted of the application of an electrical shock to the closed, or intact, and shaven chest of a dog. Special large heavy electrodes delivered 980-1,000 volts (10-12 amp.) through the chest for 0.1 second.

In those tests in which there had been a breakdown of the oxygen system and circulation for only 30 seconds, following this countershock there was invariably a satisfactory return of the blood pressure to normal, and the electrocardiogram indicated a supraventricular rhythm.

As indicated in Table 3, somewhere about 50 seconds was the dividing line between satisfactory and unsatisfactory defibrillation by the external application of a countershock in our series of experiments. This is in general agreement with the Dow and Wiggers [9] report, in 1940. Beyond this point it was found that defibrillation could always be accomplished, as indicated by the electrocardiogram (electrical defibrillation), but there was seldom return of a satisfactory blood pressure or pulse. In this situation it was found that surgical exposure of the heart, followed by a short manual pumping of the heart, quickly and successfully restored the blood pressure to a normal level. Another, and effective, method was the intra-

TABLE 2.—*Electrodes Applied Directly to Myocardium*

No. of Dogs Used	Duration of Fibrillation, Sec.	EKG Defibrillation Before Massage	Return of Circulation	Drugs	Open-Chest Massage	Then Normal B. P.
70	120	70	0	0	70	70

TABLE 3.—*Defibrillation by External Electrodes*

No. of Dog Exper.	Duration of Fibrillation, Sec.	EKG Defibrillation	Return of B. P. and Pulse	Open-Chest Massage	Then Normal Circulation
1	15	+	1	0	--
1	25	+	1	0	--
4	30	+	4	0	--
4	35	+	3	1	4
8	45	+	3	5	8
5	55	+	2	2	4
5	60	+	2	3	5
11	65	+	1	10	10
7	75	+	3	3	3
5	90	+	0	3	3
2	85	+	0	0	--
2	120	+	0	1	1

arterial perfusion of epinephrine and dextrose solution (Table 4).

In another series of 16 dogs, whose hearts were placed in ventricular fibrillation for somewhat lengthy periods, defibrillation was accomplished by an external countershock. In this group an effective circulation did not return, whereupon an intra-arterial perfusion [8] of dextrose and epinephrine was given under pressure. The latter procedure resulted in a satisfactory circulation without resort to a thoracotomy or cardiac massage.

Comment

It would appear that electrical activity, as recorded in the electrocardiogram, is not necessarily related to the effectiveness of the circulation. The addition of outside sources of electrical stimuli (pacemaker) to enhance the effectiveness of the heart beat under the condition of a cyanotic myocardium is not the simple, or final, answer. The pacemaker has found its niche in the management of the Stokes-Adams syndrome, hypothermia, and conduction disturbances resulting from septal surgery. Our results during laboratory resuscitation experiments have not indicated that it has a place in the emergency phase. For the prolonged use of the heart pacer, it may be feasible and prudent to apply one electrode to the pericardium and the other to the chest wall.

In many circumstances of acute cardiac arrest, the exact mode of stoppage, standstill, or fibrillation may not be known. The open chest, in final analysis, remains the best diagnostic procedure. However, an adequate electrical shock applied to the right upper chest near the midline and the left lower axilla will not effect cardiac standstill. This electrical shock can convert ventricular fibrillation to standstill, and if the total duration of cessation has been short, an effective beat and circulation can be accom-

TABLE 4.—*External Defibrillation and Supplemental Methods*

No. of Dog Exper.	Fibrillation Duration, Sec.	Arterial Perfusion	Arrest of Circulation, Sec.	Return of Circulation	Intravenous Medication
1	65	+	120	+	--
1	65	+	120	+	--
1	65	+	125	+	--
3	75	+	150	+	--
2	85	+	150	1	--
1	90	+	150	+	--
2	45	0	45	0	Levoarterenol
1	45	0	45	0	Epinephrine
1	55	0	55	+	Intracardiac epinephrine
1	105	+	180	0	--
1	120	+	180	+	--

plished. If the duration of fibrillation has been more than 50 seconds, in addition to the shock, a supplemental procedure must be undertaken in order to restore an effective circulation. As has been pointed out, the most valuable augmentative procedures are intra-arterial brachial perfusion of epinephrine and dextrose solution under 200 mm. Hg through a large-bore needle, or direct cardiac massage. Oxygenated blood, in place of all or a part of the dextrose solution, is efficacious. It is obvious that such a procedure cannot haphazardly be carried out and be a success. The heart that is found in a state of cardiac asystole can be reactivated to produce an effective circulation by the intra-arterial perfusion of 5% dextrose and 1-2 mg. of epinephrine.

The heart as a muscular organ will remain viable up to one hour, but in order for it to function as a hydrodynamic pump, it must have oxygenated blood in its coronary system under an adequate pressure.* The fundamental problem in cardiac resuscitation is not the method used to restart the heart but the provisio.. of a continuous supply of oxygenated blood to the brain.

Summary

In our series of dog experiments, external defibrillation with return of the circulation has a practical time limitation of about 50 seconds.

Supplemental measures to restore the circulation must be undertaken to make this procedure practical in extending the time limitation.

Intra-arterial perfusion of dextrose and epinephrine was the method of choice in these experiments.

Carnegie Medical Building, 10515 Carnegie Ave. (6).

* Forty millimeters of mercury is thought to be the minimum pressure.

REFERENCES

1. Celio, A.: Cardiac Arrest Associated with Coronary Occlusion: Successful Resuscitation, J. Internat. Coll. Surgeons 25:299 (March) 1956.

2. Beck, C. S.; Weckesser, E. C., and Barry, F. M.: Fatal Heart Attack and Successful Defibrillation, J. A. M. A. 161:434 (June 2) 1956.

3. Reagan, L. B.; Young, K. B., and Nicholson, J. W.: Ventricular Fibrillation in Patient with Probable Acute Coronary Occlusion, Surgery 39: 482 (March) 1956.

4. Mozen, H. E.; Katzman, R., and Martin, J. W.: Successful Defibrillation of the Heart: Resuscitative Procedure Started on Medical Ward and Completed in Operating Room, J. A. M. A. 162:111 (Sept. 8) 1956.

5. Kopf, E.; Zaepfel, F., and Roberts, J.: Personal communication to the authors, October, 1957.

6. Fogelson, L.: The Electrocardiogram of the Dying Heart, J. méd-biol, Moscou 6:3, 1928.

7. Negovsky, V. A.: Agonal States and Clinical Death: The Electrocardiogram During Death and Revival, Am. Rev. Soviet Med. 2:491 (Aug.) 1945.

8. Crile, G. W.: Anemia and Resuscitation, New York, D. Appleton & Company, 1914.

9. Dow, P., and Wiggers, C. J.: Limitations of Myocardial Recovery from Ventricular Fibrillation Through Countershock, Proc. Soc. Exper. Biol. & Med. 45:355 (Oct.) 1940.

10. d'Halluin, M.: Résurrection du cœur, la vie du cœur isolé, le massage du cœur, Paris, Vigot frères, 1904.

A Suction Apparatus for Use During Open Cardiotomy

ELTON WATKINS JR., M.D., Boston, and COMDR. ALEXANDER C. HERING (MC), U. S. N.
With the Technical Assistance of Mary Ann Dalton, R.N., and Ralph W. Sommers

There are certain trying moments during open-heart surgery. At these times a high-volume suction apparatus is necessary to remove large volumes of blood from the operative field and to provide a means for accurate measurement of this loss so that correct transfusion replacement may be instituted promptly. The suction pumps of the modern pump-oxygenator should be supplemented by such a unit to remove blood generally from the thoracotomy wound. Conventional operating-room suction is not ideal for this purpose: The suction container is usually placed beneath the operating table or on the floor, where it is not easily observed by personnel in charge of blood replacement; the suction bottles are usually large and wide, so that it is impossible to measure the volume of aspirated loss accurately, and autotransfusion of the aspirated blood is difficult.

With these objections in mind, we have developed a compact suction unit which is siliconized and may be autoclaved for purposes of aspiration during open cardiotomy.* The apparatus consists of two long, thin glass Kelley flasks, of 900-ml. capacity, and a special valve manifold which permits connection of the bottles to the general operating-room suction and to the patient (Fig. 1). The channels in the valve manifold are arranged in such a manner that either of the narrow collection flasks may be used for the collection of blood or may be emptied into the general operating-room suction container without interruption of continuous suction at the operating field. The flasks and the valve manifold are treated with silicone to minimize blood trauma and coagulation. Tygon tubing connects the various channels so that the entire unit may be completely dismantled for cleaning and steam sterilization.

By using two thin flasks and a valve, it is possible to record and then discard high volume losses rapidly and accurately, or to collect blood contaminated with citrate or other chemicals in one of the flasks, so that the contents of the other container are kept uncontaminated. Autotransfusion into the pump may be accomplished rapidly by clamping the anterior suction line at the valve manifold, turning the valve lever to evacuation position and draining blood from the bottom outlet of the Kelley flask into the pump.

Description of the Apparatus

The Valve Manifold.—The connections of the bottles to the various channels of the valve manifold are diagrammed in Figures 2 and 3. With the valve lever in the upper position on left or right, either the left or the right flask is connected to suction and

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From the Department of Surgery, the Lahey Clinic, Boston, and the Surgical Research Laboratory, U. S. Naval Hospital, Chelsea, Mass.

The opinions or assertions contained herein are the private ones of the writers, and are not to be construed as official or reflecting the views of the Navy or the Naval Service at large.

Department of Surgery, the Lahey Clinic; Consultant in Surgery and Physiology, U. S. Naval Hospital, Chelsea, Mass. (Dr. Watkins). Chief of Thoracic Surgery, U. S. Naval Hospital, Chelsea, Mass. (Commander Hering).

* Manufactured by Edward A. Olson, Ashland, Mass.

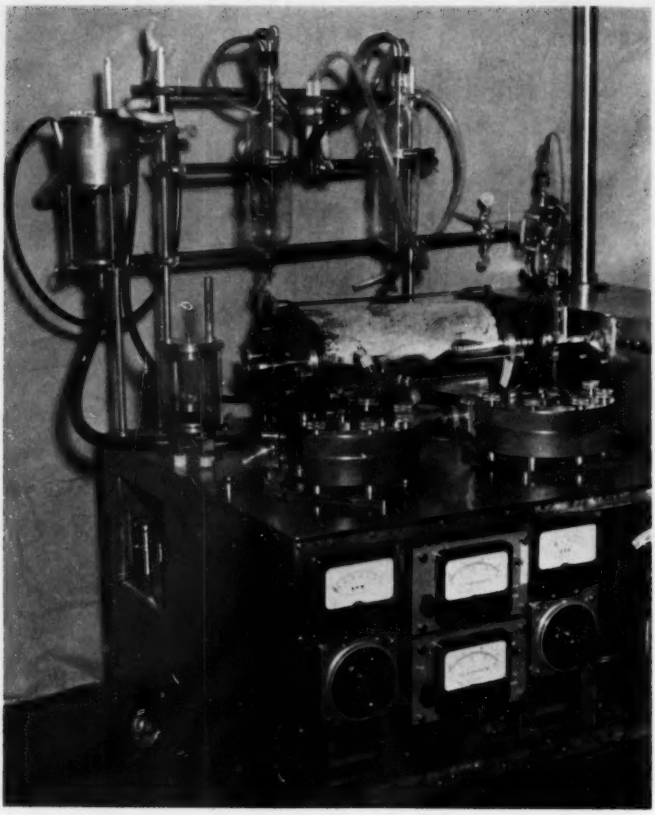


Fig. 1.—General view of suction unit on a metal frame mounted at the rear of our pump-oxygenator.

to the operative-field line so that blood lost in the operative field empties into that flask. When the indicator lever is moved to the lower position on either side, that flask empties into the general discard, while suction is automatically transferred to the contralateral bottle during the period of emptying—hence there is no interruption of suction at the operating field.

The core of the valve is fashioned of stainless steel Type 17-4PH, while the barrel is made of Type 303 stainless steel. The use of different grades of steel in this manner minimizes the binding effect which occurs in contacts of uniform-grade steel. The valve parts are treated with a silicone resin.† Our experience with this silicone resin indicates that a number of precautions must be taken during its application. The part to be treated should be preheated for a period of 15 to 20 minutes in a low-temperature oven to eliminate surface moisture. After cooling of the part in a dry atmosphere, the resin is applied by dipping, taking care that no bubbles are apparent on the surface of the resin solution or during withdrawal of the part from immersion. The treated apparatus is then air-dried for one hour and baked for four hours at a temperature of 400 F in a kitchen oven. The nylon connectors of the valve manifold are removed during silicone treatment. Prior to infrequent retreatment with the resin, residual silicone may be removed by scrubbing with xylene. Before use, contact-

† Resin R671, Lot 744, Dow Corning Company, Midland, Mich.

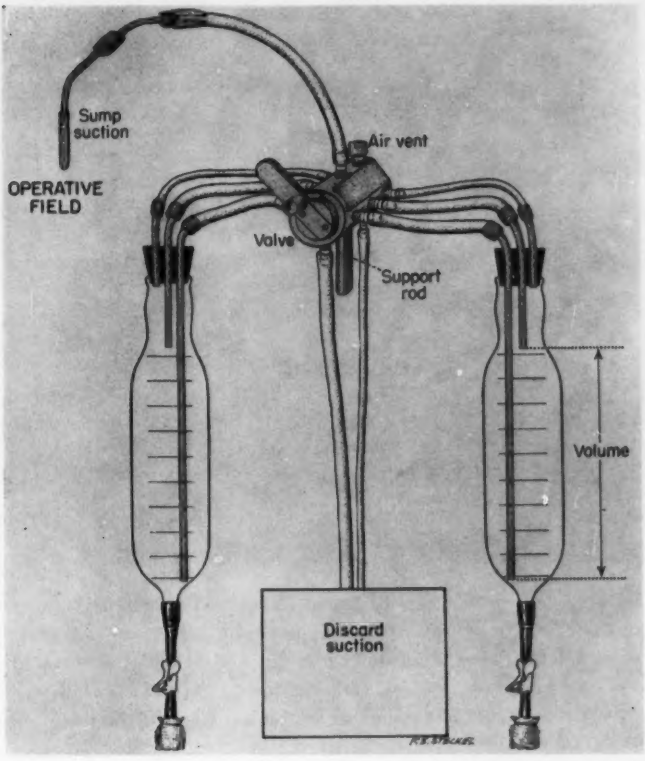
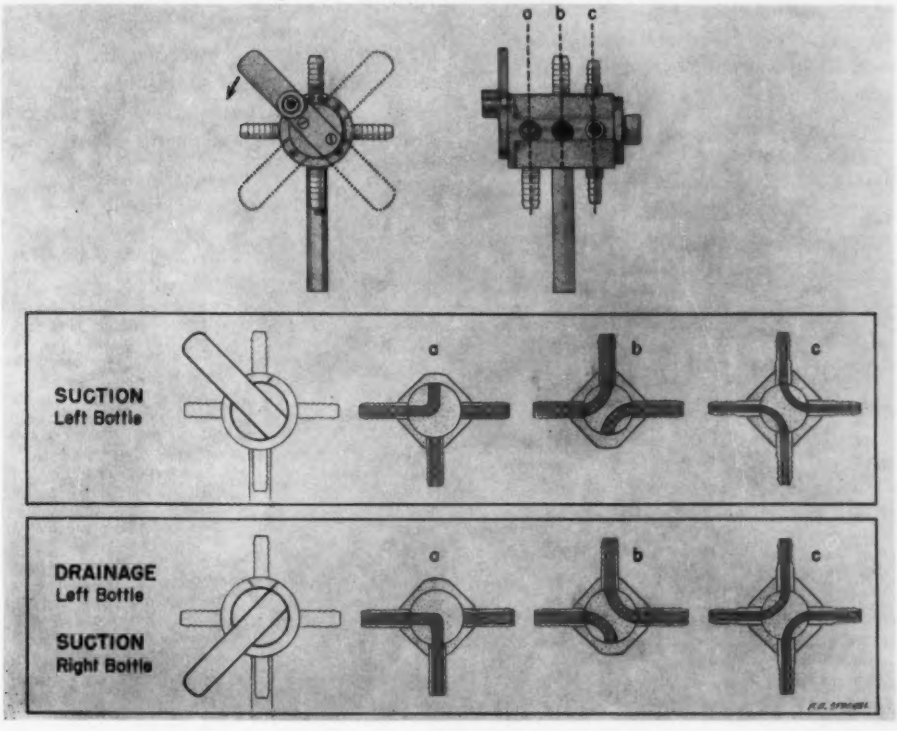


Fig. 2.—Method of connection of valve outlets to aspiration flask.

Fig. 3.—Channels within the valve manifold.



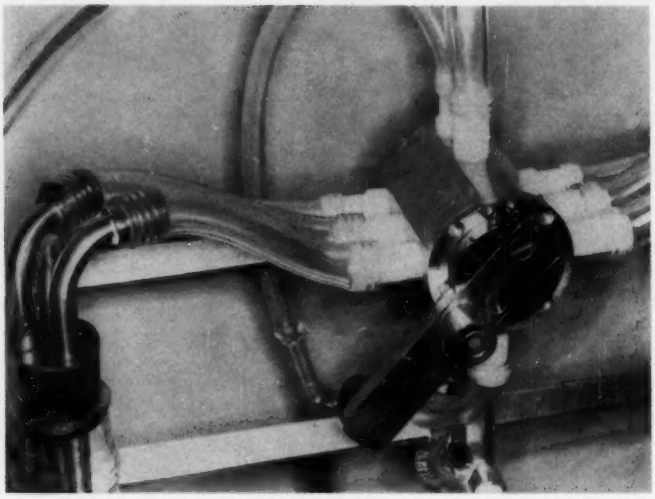


Fig. 4.—Connection of valve outlets to flask with Tygon tubing.

ing valve surfaces are lightly coated with silicone high-vacuum grease.‡

Collection Flasks and Tubing.—The glass vessels are 900 ml. Kelley flasks.§ The flasks are calibrated so that minute-to-minute volumes may be observed and recorded. Stainless-steel tubes are inserted into each flask through a three-hole rubber stopper. The flasks and steel tubes are treated with silicone resin in a manner similar to treatment of the valve manifold. The ends of the steel tubes are flared to connect to B-44-3 Tygon tubing,‖ having an inner diameter of $\frac{3}{8}$ in. and a wall thickness of $\frac{3}{32}$ in. The channels for conduction of suction and air in the last stage of the manifold are segments of B-44-3 tubing, with an inner diameter of $\frac{1}{4}$ in. and a wall thickness of $\frac{1}{16}$ in.

Rubber tubing is used to connect the valve-manifold drainage outlets to the discard suction bottle. The sump-suction aspirator tip is black, to distinguish it in the op-

‡ Dow Corning Company, Midland, Mich.

§ Macalaster Bicknell Company, Cambridge 38, Mass.

‖ U. S. Stoneware Company, Akron, Ohio.

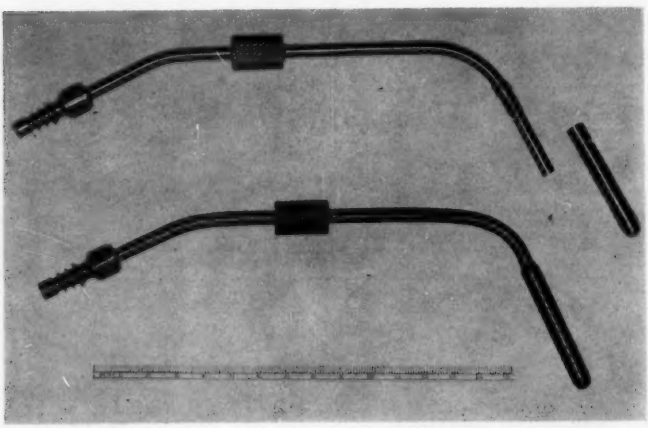


Fig. 5.—Sump aspirators used in the operative field. (Aspirators are of the type originally developed at the Childrens' Medical Center, Boston.) One aspirator has a black finish (bottom unit) to identify it immediately as being attached to the apparatus described here for general thoracotomy suction. Two aspirators of similar design, but with a natural metal finish (top unit, dismantled), are connected to the two channels of the suction pump on our oxygenator for purposes of immediately returning to the oxygenator circuit any blood aspirated from within the opened heart.

erative field from two similar sump-suction aspirators connected to the De Bakey suction pump of the oxygenator (Fig. 5). The aspirator tip is connected to the inlet of the valve manifold with Tygon tubing of $\frac{3}{8}$-in. inner diameter and $\frac{3}{32}$-in. wall thickness.

The various parts of the device are secured with clamps on a horizontal stainless-steel bar. The bar is rectangular in cross section so that the clamps will not twist from position. The apparatus is sterilized, assembled as a unit, and clamped to a bar frame on our pump-oxygenator, being first used during the period of bypass (Fig. 1). After bypass, the apparatus on its bar is detached from the oxygenator as a unit and secured by clamps to the edge of the operating table so that the oxygenator may be moved for cleaning.

Summary

A suction apparatus has been developed specifically for use during open-heart surgery. Valving connections of the unit allow continuous suction into narrow flasks for accurate fluid volume measurement. The unit is attached to a conspicuous place on our pump-oxygenator, so that losses are immediately apparent to pump technicians. Provision is made for transfer into the pump-oxygenator of blood collected in the sterile siliconized chambers of the apparatus.

Lahey Clinic, 605 Commonwealth Ave. (14).

Idiopathic Hyperlipemia

Special Reference to Abdominal and Coronary Symptoms

MAJOR RAIMONDS J. GAILITIS (MC), U. S. Army, and WERNER SCHREIBER, Dr. Med., Nürnberg, Germany

Idiopathic hyperlipemia represents the homozygous abnormal state. The basic metabolic defect in this condition is a retarded removal of ingested fat from the blood stream after normal absorption. The homozygous abnormal state is further complicated by hepatosplenomegaly; abdominal, coronary, and central nervous system crises, and, frequently, secondary xanthoma. Many patients believed to be "psychotic" proved to be subject to idiopathic hyperlipemia after thorough diagnostic screening. Furthermore, many patients seen in the emergency room because of an acute abdominal crisis, and subjected to a number of surgical procedures, often are helped as a result of a simple observation of "milky serum," which they exhibit during these acute episodes. These manifestations are believed to be due to an increase in neutral fats in particulate form, which impart a milky turbidity to the serum, known as lactescence. This milky appearance becomes evident if the lipid level reaches about 4,000 mg. %. There is also often a concomitant, but less pronounced, rise in serum cholesterol and phospholipid levels.[8] Low-density lipoprotein classes are more elevated than the high-density ones. Transient hyperglycemia and decrease in serum lipase levels have been encountered frequently. Klatskin and Gordon [8] suggested that the abdominal crises and other symptoms encountered in this disorder may be due to embolization of agglutinated serum lipid particles in the corresponding organs. The liver and spleen may be enlarged, and this may account for abdominal pain; however, this phenomenon is observed more frequently among children, whereas in adults hepatosplenomegaly is not observed as often.[1]

Three types of hyperlipemia are recognized.

1. Alimentary hyperlipemia, in which neutral fat reaches the peak four to six hours after the ingestion of a fat-rich meal. (There is no increase in the cholesterol or phospholipids in this type of hyperlipemia.)
2. Secondary hyperlipemia, observed in patients with diabetic acidosis, nephrosis, pancreatitis, myxedema, starvation, von Gierke's disease, Niemann-Pick disease, pregnancy, fetal hyperlipemia, hyperlipemia in anemias, amyloidosis, renal vein thrombosis, and toxic hepatosis
3. Idiopathic hyperlipemia

Report of Cases

CASE 1.—A man, aged 35, was admitted to Grant Hospital, Chicago, on Sept. 12, 1955. He had had very sudden severe epigastric pain, which had its onset about one and one-half hours following eating of eggs, sausage, milk, and butter. In several hours the pain spread throughout his abdomen and made him "double up" and vomit. He experienced many dramatic episodes of precordial pain and dyspnea in the next three hours. Morphine sulfate, 15 mg. I. M., failed to improve his symptoms. The patient had had at least seven similar episodes previously. These also had followed rather heavy meals. Upper gastrointestinal and gallbladder series on three separate occasions had failed to reveal any pathology. The patient had no dietary management and made an uneventful recovery after a three- to four-day stay at home on a low-caloric intake. These episodes were accompanied by high temperatures. The patient has three sisters, all of whom are well, and cholesterol levels determined on all of them were within normal limits.

Physical Examination.—The patient was in very acute distress. The skin was pale and covered with perspiration. Temperature 101.6 F; pulse rate 100 per minute; blood pressure 150/80 mm. Hg. The EKG revealed marked ST depression in

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U. S. Army Hospital, Nürnberg, Germany. Chief of Gastroenterology Section (Major Gailitis).

Leads II, III, aVF, V5, and V6, with negative T-waves. No icterus was present. The neck was supple. The ocular fundi were very pale. Crackling rales were present in both lung bases. There were no cardiac murmurs. The abdomen was distended. The patient avoided deep breathing, and his abdominal muscles were very rigid. Marked tenderness was present in the upper epigastrium. The liver was tender and slightly enlarged, and some ascites was present. A flat plate of the abdomen also suggested an increase in abdominal fluid. On withdrawing venous blood, it was noted that the blood had milky appearance, with total lipids of 4,385 mg. %, cholesterol 363 mg. %, serum amylase 138 units, blood sugar 210 mg. %, alkaline phosphatase 4.9 Bodansky units, and blood lipase 0.6 unit. Urinalysis showed 1+ albumin and 3+ sugar. The patient was receiving intravenous dextrose while these tests were being done.

On the second hospital day (Sept. 13) the patient had severer abdominal pains, looked worse, and was maintained on intravenous fluids only, with 500 mg. tetracycline (Achromycin) b. i. d. The cholesterol level dropped to 305 mg. %; amylase, 78 units, and glucose, 175 mg. %. Temperature was 101.0 F. Throughout the first three days the patient was comfortable only on morphine and chlorpromazine (Thorazine) every three hours. Symptoms were improved, with a total lipid level of 1,230 mg. % five days later. On the sixth day of hospitalization the patient was started on a 25 gm. vegetable-fat diet. He was discharged on this regimen.

Six months later his total lipids were 386 mg. %. The urine was normal. The patient has remained in good health and has not had any discomfort whatever.

CASE 2.—A white woman aged 36, unipara, primigravida, was admitted to the 20th Station Hospital, Nürnberg, in March, 1957. She was subject to very severe epigastric and midabdominal pains, of sudden onset. Nausea and vomiting followed, and she was considered to be seriously ill. The pains assumed a more constant character at the time of admission and involved practically the entire abdomen. The past history revealed that she had had many similar episodes of abdominal pain. The attack would start with rather dramatic suddenness. A weight loss of about 26 lb. was reported. As a result of these symptoms, a diagnosis of acute colitis was made in 1947, and cholecystitis was suspected on several occasions subsequently. In 1949 an exploration was done, and the left ovary was removed because of "cystic changes." In 1951 hysterectomy was performed because of irregular bleeding and pains. In 1953 the right ovary was removed. The family history revealed that one sister and one brother had "gallbladder symptoms." Their cholesterol levels were 280 and 290 mg. %, respectively.

On physical examination, the patient appeared to be in very acute distress. The temperature was 102.4 F; respirations were 28 per minute. The lungs were clear. Heart sounds were of good quality, and no murmurs were present. Blood pressure 125/80 mm. Hg; pulse rate 102 per minute. The abdomen was very tender over the epigastric and umbilical areas. The liver was slightly enlarged and tender.

X-ray examination revealed some evidence of paralytic ileus. The chest x-ray was normal. An EKG showed nonspecific ST changes in Leads I and aVL. Serum amylase was 32 units; white cells 12,100 per cubic millimeter; differential count normal. Urinalysis was normal. A gallbladder series revealed a nonfunctioning gallbladder. The patient was given meperidine (Demerol) and intravenous fluids and was discharged as improved with the diagnosis of acute cholecystitis and paralytic ileus. Upper gastrointestinal and gallbladder x-rays were normal several weeks later. The patient continued to have abdominal pain despite the management on a "low-fat" diet, and she needed rather liberal doses of Donnatal.*

Idiopathic hyperlipemia was suspected and verified by examination of a blood specimen, which showed total lipids to be markedly elevated (2,342 mg. %). Serum lipase was 0.6 unit; serum amylase 34 units; phospholipids 142 mg. %. The patient was given a 25 gm. vegetable-fat diet. She experienced rather dramatic improvement and has remained symptom-free since.

Four weeks later the total lipids were normal (632 mg. %); cholesterol was 210 mg. %.

CASE 3.—A 46-year-old white man was admitted to the 20th Station Hospital, Nürnberg, because of very severe abdominal pain. The patient told of having had a feverish feeling, general aching, tiredness and retrosternal pain for at least 12 years. Symptoms had lately been getting almost intolerable. A number of x-rays had been taken six months before during a hospital admission; however, they were all normal. An EKG revealed only nonspecific ST changes in Leads I, II, III, and aVF, although myocardial infarct was suspected on one occasion. It was learned that the patient had been studied in mental institutions in 1954. One day following the present admission the patient appeared to be very disturbed and wanted to make out "his last will." He told us that he was suffering from cancer. The psychiatrist called him markedly "anxious and paranoid" at this time.

Physical examination revealed the patient to be in very acute abdominal pain, pulling up his knees at times. The fundi were pale. The lungs were

*An antispasmodic sedative, containing phenobarbital and belladonna alkaloids.

clear. Heart sounds were of good quality. The abdomen was very distended. The liver and spleen were moderately enlarged and tender.

Blood withdrawn was noted to be almost white. Total proteins were 6.5 gm. %, with albumin 2.7 gm. % and globulin 3.8 gm. %. Total lipids were 4,182 mg. %; phospholipids, 915 mg. %; cholesterol 681 gm. %; serum amylase, 8 units; serum lipase, 0.1 unit. Red blood cell count was 3,700,000; sedimentation rate 22 mm. in one hour; white cell count 13,850. Calcium was 8.9 mg. %; K^+ 4.0 mEq/L.; chlorides, 92.3 mEq/L. Urinalysis was normal. Blood sugar was 116.5 mg. %; the sulfobromophthalein (Bromsulphalein; BSP) test gave 40% dye retention in 45 minutes.

The patient made uneventful recovery on intravenous fluids only. He lost more than 20 lb. in several days and remained perfectly well on a 20 gm. vegetable-fat diet. His serum lost its lactescence in about three days. Eight days later he was given permission to visit his family; however, he returned to the hospital 36 hours later with moderately severe abdominal pains and distention. He has been managed on a 20 gm. vegetable-fat and has remained in fine condition. Complete chemical and electrophoretic studies done on three of his daughters (aged 4, 6, and 8 years) were within normal limits.

CASE 4.—A 34-year-old woman was examined in September, 1957. She was subject to very severe abdominal cramps following fatty meals. Abdominal cramps were accompanied by nausea and vomiting and lasted several days. The patient had a normal gallbladder and upper and lower gastrointestinal series. Symptoms had been present for at least eight years; however, they were interpreted as "recurrent gallbladder" attacks. She had been hospitalized at least seven times and was subjected to bladder-neck dilations because of the stricture. Chloramphenicol (Chloromycetin) and nitrofurantoin (Furadantin) were required in treating her "phlegmonous cystitis" on two occasions. The patient was seen several times every week in the emergency room for her epigastric symptoms.

On July 11, 1958, chemical analysis for total lipids revealed 2,067 mg. %, and idiopathic hyperlipemia was then suspected. She was given a 20 gm. vegetable-fat diet, with very dramatic improvement. Repeated total lipid determinations now revealed levels ranging from 400 to 800 mg. %. The patient has remained in good health (Table).

Comment

Four cases of idiopathic hyperlipemia are presented. Diagnosis, emergency care, and treatment are discussed. All patients made an uneventful recovery and remained well on a very strict 20-25 gm. vegetable-fat diet. The first patient represented a case of long-lasting disease, characterized by dramatic abdominal crises. In the first three cases the electrocardiogram revealed nonspecific ST changes. In the second case the diagnosis was never suspected and three surgical interventions were carried out. Initial relief was achieved by intramuscular morphine and chlorpromazine in three hours, and constant intravenous fluids were required to produce permanent absence of symptoms. The third patient showed dramatic recurrence of symptoms during his early return to a more liberal fat intake. It has been shown by many observers that, to some extent at least, the low-fat diet may decrease the degree of hyperlipemia.[2] This treatment is not feasible at all times, since the patient may very easily neglect the planned regimen. In these instances drug therapy may be of some help. Heparin, for instance, produces decrease in the serum turbidity and also produces an absolute decrease in fat contents.[6,7] At the same time, a shift toward lipoproteins of higher density occurs. Heparin medication is occasionally used in cases of hyperlipemic crisis. Heparin apparently acts as an activator of a tissue factor to produce a lipemia-clearing factor. The remarkable effect of chlorpromazine on blood was demonstrated by Hollister and associates.[7] They were able to produce a clearing of the patient's serum

Data on Four Patients with Idiopathic Hyperlipemia

Case	Total Lipids, Mg. %	Cholesterols, Mg. %	Phospholipids, Mg. %	Lipase, Units	Serum Amylase, Units	Neutral Fat (Triglycerides), Mg. %
1	4,385	363	--	0.6	138	3.640
2	2,342	310	142	0.6	34	1.860
3	4,182	681	915	0.1	8	3.680
4	2,067	155	62	0.2	8	1.720

following the administration of chlorpromazine. The action of chlorpromazine is possibly mediated by its adrenolytic activity. Klatskin and Gordon [8] reported that the administration of corticotropin increased the turbidity of the serum, with some increase in the total fatty acids. This suggested that corticotropin may produce turbidity by altering the physical state of the lipids. Rich and his co-workers [9] noted that cortisone caused a rise in the serum lipids. Hydrocortisone has an opposite metabolic effect. The role of adrenal steroids in the management of essential hyperlipemia is not widely accepted.

Estrogens.—As Robinson et al.[10] have pointed out, there is a direct relationship between serum lipids and estrogen metabolism. Estrogens could be used preferably with women in the treatment of hyperlipemia, because of fewer side-effects. In the male patients these side-effects of estrogen treatment would produce feminization with gynecomastia and testicular atrophy.

Nicotinic Acid.—Altschule et al. investigated effects of nicotinic acid on the serum lipids. They proved that nicotinic acid lowers the serum cholesterol level and that this action might be twofold. First, nicotinic acid might increase phagocytosis of cholesterol by the reticuloendothelial system. Second, nicotinic acid might have an indirect action through its effects on the lipids, such as neutral fats, by which action it would protect the liver in a manner similar to that of lipotropic factors. How far disturbed tyrosine metabolism in the nicotinic-acid deficiency state (pellagra) affects thyroxin production, and whether it is the deficiency of nicotinic acid that is primarily responsible for the serum cholesterol in hyperthyroidism, is unknown. However, thyroid hormone has very profound effect on the serum cholesterol and lipids, causing a decrease in their concentrations.

Sitosterol.—The effects of this sterol on serum lipids have been studied, and favorable results have been obtained in atherosclerosis; however, these effects were only transitory.

The control of idiopathic hyperlipemia is a matter of a long-term management and requires the acceptance of, preferably, a 20 gm. vegetable-fat diet by the patient concerned. In our four cases this long-term treatment has resulted in the abandonment of all drugs used by the patient. It seems that a good cooperation by the patients is an answer to the management of idiopathic hyperlipemia.

Summary

Four cases of idiopathic hyperlipemia are added to the literature. Many surgeons, internists, and practitioners have been consulted during the numerous hospital admissions these patients were subject to.

Failure to recognize this condition might have resulted in unnecessary surgery. Laboratory tests short of total lipid level determination are not adequate for the diagnosis of idiopathic hyperlipemia. As x-ray, laboratory, and clinical examinations were not characteristic of gallbladder disease, pancreatitis, or perforated viscus, electrocardiography was resorted to, to rule out an atypical coronary disease. Finally, these patients were treated symptomatically with intravenous fluids and meperidine (Demerol), and eventually got better temporarily. All of them returned to the hospital for diagnostic work-up and symptomatic management. One of them had had three laparotomies, and one was treated in an institution for "psychosis." All four of these patients presented now enjoy good health and are maintained on about a 25 gm. vegetable-fat diet.

U. S. Army Hospital, Nürnberg, Germany; APO 696, New York.

REFERENCES

1. Bernstein, S. S.; Williams, H. H.; Hummel, F. C.; Shepherd, M. L., and Erickson, B. N.: Metabolic Observations on a Child with Essential Hyperlipemia, J. Pediat. 14:570, 1939.

2. Bloomfield, A. L., and Shenson, B.: Syndrome of Idiopathic Hyperlipemia with Crisis of Violent Abdominal Pain, Stanford M. Bull. 5:185, 1947.

3. Corazza, L. J., and Myerson, R. M.: Essential Hyperlipemia: Report of 4 Cases, with Special Reference to Abdominal Crisis, Am. J. Med. 22:258, 1957.

4. Furman, R. H.; Howard, R. P.; Norcia, L. N., and Keaty, E. C.: Influence of Androgens, Estrogens and Related Steroids on Serum Lipids and Lipoproteins, Am. J. Med. 24:80, 1958.

5. Gordon, R. S., Jr., and Cherkes, A.: Unesterified Fatty Acid in Human Blood Plasma, J. Clin. Invest. 35:206, 1956.

6. Herzstein, J.; Wang, C. I., and Adlersberg, D.: Effect of Heparin on Plasma Lipid Partition in Man: Studies in Normal Persons and in Patients with Coronary Atherosclerosis, Nephrosis and Primary Hyperlipemia, Ann. Int. Med. 40:290, 1954.

7. Hollister, L. E., and Kanter, S. L.: Essential Hyperlipemia Treated with Heparin and Chlorpromazine, Gastroenterology 29:1069, 1955.

8. Klatskin, G., and Gordon, M.: Relationship Between Relapsing Pancreatitis and Essential Hyperlipemia, Am. J. Med. 12:3, 1952.

9. Rich, A. R.; Cochran, T. H., and McGoon, D. C.: Marked Hyperlipemia Resulting from Administration of Cortisone, Bull. Johns Hopkins Hosp. 88:101, 1951.

10. Robinson, R. W.; Higano, N.; Cohen, W. D.; Sniffen, R. C., and Sherer, J. W.: Effects of Estrogen Therapy on Hormonal Functions and Serum Lipids, in Men with Coronary Atherosclerosis, Circulation 14:365, 1956.

11. Zarafonetis, C. J. D.; Miller, G. M.; Seifter, J.; Baeder, D.; Myerson, R. M., and Steiger, W. A.: Metabolic Studies in Patient Receiving Lipid Mobilizer Hormone, Am. J. M. Sc. 234:493, 1957.

Tracheotomy in Burns

JOHN A. MONCRIEF, M.D., Atlanta

The therapeutic and prophylactic use of tracheotomy in crushing injuries of the chest and in cases of poliomyelitis and tetanus in which respiratory difficulties or complications require diminution of the dead space of the tracheobronchial tree is well established as an important part of the patient's treatment. The use of a tracheotomy to bypass acute or chronic obstructions of the larynx and as a prophylactic procedure associated with many cases of head and neck surgery is an established fact. However, the use of a tracheotomy in the treatment of thermal injury as either a prophylactic or a therapeutic procedure has not met with widespread acceptance, and there is still considerable disagreement as to both its need and its value.

Thermal injury to the respiratory tract below the larynx is uncommonly seen even in extensive burns and is difficult to produce experimentally in the absence of actual burning of the nasopharynx or posterior pharynx. Exposure of the glottis to heat causes a reflex closure of the glottis and thus protects the lower respiratory tract. Hot air is cooled with extreme rapidity, and has relatively little heat-carrying capacity, and therefore its ability to cause a burn of the cooled moist surface of the respiratory tract is minimal. However, steam, with a tremendously high heat-carrying capacity, can easily cause a true thermal burn of the respiratory tract if in some manner this superheated vapor can get beyond the glottis. Such a circumstance, however, would be unusual.

When a person is subject to a thermal injury while in a confined area, however, he may be forced to inhale irritant smoke and gases. Although he may not sustain any direct thermal injury to the respiratory tract, the irritation and inflammation attendant upon exposure to these irritants frequently result in functional impairment of great significance. The edema of the tracheobronchial mucosa and the outpouring of protein-rich exudate, which obstruct the alveoli and terminal bronchioles, mechanically interfere with the exchange of air in the respiratory tree, and purely on the basis of mechanical interference from pulmonary edema the patient dies of suffocation.

This irritant inflammation of the tracheobronchial tree is seen to varying degrees in many cases of thermal injury and is of great significance in the immediate postburn period.

A second- or third-degree burn of the face and neck results in the rapid appearance of massive edema of the burn area. This edema not only closes the eyes but everts the lips and immobilizes the jaws to such an extent that the mouth, if it can be opened at all, is opened with great difficulty. Under these circumstances the nausea and vomiting which is prone to occur in burns of over 20% may easily result in the aspiration of gastric contents into the respiratory tract. The edema of the neck, although more obvious externally, also involves the deep tissues of the neck and the posterior pharynx, making breathing and swallowing more difficult. Similarly, coughing and clearing of the tracheobronchial tree of mucus, which is present in increasing amounts, are made more and more difficult.

In the presence of massive edema of the face and neck, general anesthesia is extremely difficult from the standpoint of establishing and maintaining an adequate airway. After resolution of this edema, which then diminishes the airway difficulty,

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one meets the problem of traumatizing the tissues of the face with an anesthesia mask to such an extent that conversion of partial-thickness burns to full-thickness skin loss frequently occurs, and not infrequently severe deformities result. With deep burns of the face requiring repeated grafting procedures and reconstructive surgery, it is very difficult without the aid of a tracheotomy to assure an adequate airway during the operative procedure and during the postoperative period, when large pressure dressings are in place.

The burned patient, because of the location or extent of his burns, is frequently immobilized in such a manner that clearing the tracheobronchial tree of even the normal amounts of secretions which are present is frequently difficult, and if there is an increased amount of secretions this difficulty is multiplied. During the early postburn period, particularly during episodes of septicemia, the patient may be so ill that clearing the tracheobronchial tree by the normal mechanism of coughing is not effectively carried out, and therefore aspiration of the tracheobronchial tree to stimulate coughing and remove copious or tenacious secretions is mandatory. Certainly this is most easily accomplished through a tracheotomy.

In view of these problems involving the tracheobronchial tree attendant upon thermal injury, it is felt that a review of the experiences of the U. S. Army Surgical Research Unit, Brooke Army Medical Center, Fort Sam Houston, Texas, would be of value. During the period of 1950 through November, 1956, over 1,000 burn cases have been treated, and these have been reviewed from the standpoint of the application of tracheotomy as a prophylactic and therapeutic tool in an attempt to minimize the complications which may result from burns. Eighty-four patients have had a tracheotomy performed during the period 1950 through October, 1956, for the indications shown. No instance of a true thermal burn of the tracheobronchial tree has been noted, and this is due to the fact that during this period no steam burns except those of very minor extent on peripheral portions of the body were encountered. The indications for tracheotomy have been divided into therapeutic and prophylactic indications, and these are defined in the following section.

Therapeutic Indications

Respiratory Irritation.—Evidence in the immediate postburn period of edema of the tracheobronchial tree and increased secretions as a result of sustaining the thermal injury while in a confined space, resulting in the inhalation of irritant smoke and gases.

Respiratory Embarrassment.—Mechanical impairment of the passage of air due to local edema of the larynx without edema of the tracheobronchial tree, or secretional obstruction of the tracheobronchial tree due to copious or tenacious secretions which the patient is unable to clear by his own cough mechanism.

Associated Injury.—This has been seen in only one patient, who had multiple rib fractures and a hemopneumothorax, and tracheotomy was performed, not on the basis of the burn, but because of the chest injury itself.

Prophylactic Indications

Tracheobronchial Toilet.—In those patients in whom there is an inability to clear even the normal amount of tracheobronchial secretions because of immobilization, location of the burn, extent of the burn, or poor cooperation, tracheotomy is done as a prophylactic measure to prevent obstruction of portions of the tracheobronchial tree.

Anesthesia.—In those patients with extensive face burns from whom it is anticipated that repeated general anesthetics will be necessary, it has been felt that a tracheotomy is much less traumatic than repeated endotracheal intubation, which is necessary if one is to prevent traumatizing the burned face with an anesthesia mask.

Deep Face Burns.—In patients with deep burns of the face, the marked edema which occurs not only in the face but in the tissues

of the neck and posterior pharynx exposes the patient to the danger of aspiration of gastric contents if vomiting occurs and decreases the patient's ability to clear his tracheobronchial tree with his normal coughing mechanism, and the subsequent course of therapy with repeated anesthetic episodes is facilitated by a tracheotomy.

Of the 84 patients having tracheotomies, only 4 did not have burns about the face. All four of these burns were rather extensive, and the difficulty with clearing of secretions from the tracheobronchial tree occurred at varying periods after the burn. This complication developed not as a result of localized burn about the face and neck but as an accompaniment of the general deterioration of the patient's condition in the immediate postburn period or during the later stages, when other complications developed.

Of the 84 patients having tracheotomy, 48 died. Of these 48 deaths, only 5 were respiratory deaths. The majority of the patients died as a result of septicemia, which in itself is an indication for tracheotomy only if the patient's general condition has deteriorated to such an extent that he is unable to clear his tracheobronchial tree adequately. None of the 14 patients on whom tracheotomy was done for respiratory irritation or respiratory embarrassment survived, and of this total of 14 patients, 5 died as a direct result of respiratory mechanical obstruction and 4 died of pulmonary complications associated with their burn and their burn therapy. The other patients on whom tracheotomy was done as a prophylactic procedure had a much more favorable outcome. Although 45% of these persons died, none of them did so as a result of respiratory complications, and the majority of the deaths were due to septicemia.

In the 10 patients in whom the tracheotomy was done as an emergency procedure, usually on the ward, only one survived. However, of those 10 patients, the emergency tracheotomy appeared to be of immediate value in 4; and, although only 1 of these 4 survived, 2 of the deaths were due to septicemia and 1 to pulmonary edema following the initial relief from the respiratory embarrassment.

Comment

Tracheotomy was performed on a total of 84 persons from 1950 through October, 1956. Of these 84 patients, 48 died, but only 5 deaths were attributed to respiratory obstruction or inflammation. Death in the vast majority of cases was due to septicemia.

Tracheotomy was considered necessary as a therapeutic procedure for 14 patients. Because of edema of the tracheobronchial mucosa or secretional obstruction, none of them survived. Four of these patients died as a result of pulmonary edema, and four died of asphyxiation due to edema of the bronchial mucosa and secretions that could not be cleared. One patient, who was previously relieved by a tracheotomy, died as a result of a laryngeal obstruction caused by improper placement of a tracheotomy tube that slipped out.

In instances in which a therapeutic tracheotomy was indicated, the patients appeared to derive little benefit from this procedure except when localized obstruction occurred in the region of the larynx. It does not seem logical to expect any benefit from a tracheotomy tube if inhalation of irritating gases or smoke has caused extensive edema of the tracheobronchial mucosa. However, a patient might receive considerable benefit upon the clearing away of copious or thick secretions from the tracheobronchial tree, thus stimulating coughing.

Only one of the patients on whom tracheotomy was performed as a therapeutic procedure for respiratory embarrassment due to mechanical or secretional obstruction died of a cause considered respiratory, i. e., pulmonary edema. Although six of the seven patients died on whom a prophylactic tracheotomy was performed for tracheobronchial toilet, not one of these six patients died as a result of respiratory complications.

Initial appraisal of the results of emergency tracheotomy is disappointing. Of 10 cases, only 1 patient survived; nevertheless, immediate relief was apparent in 4 instances, including the patient who survived. As an emergency procedure, the tracheotomy fulfilled its intended purpose, since two of the three deaths were caused by septicemia many days after the tracheotomy was performed, and the other death was caused by pulmonary edema four days after the operation.

From Jan. 1, 1955, through October, 1956, a total of 58 patients sustaining burns of the face did not undergo a tracheotomy. There were 47 survivors and 11 deaths in this group, and not a single death was attributed to a respiratory cause. It might be inferred that tracheotomy was of little value, since the survival rate was much higher in the group of patients on whom a tracheotomy was not performed. However, the group that did not receive this therapeutic procedure had burns of a much less extensive area, covering not more than one-half the face. The burns of the face and neck in this group were not as deep as those in the group that had a tracheotomy, and the burns of the neck were not extensive. As a result, less edema of the face and neck occurred, and usually obstruction of oral and respiratory passages was insignificant. For these reasons, a tracheotomy was not indicated.

Some unfortunate complications have occurred in the management of tracheotomy. The tracheotomy tube placed in one patient was barely long enough to reach into the trachea; as a result, the tube slipped out into the soft tissue of the neck when the patient was turned face down on the Stryker frame. This patient died of asphyxiation, caused by obstructing edema of the larynx above the tracheotomy opening. In another instance, a perforation of the trachea resulted from using a rigid metal tracheotomy tube that was too long. This condition was found at autopsy, and, although it was not considered the cause of death, undoubtedly it would have caused serious complications had the patient survived for a longer period. In another instance, the trauma of the tube resulted in severe erosion of the trachea.

The tracheotomy tube used for a child was of smaller caliber but of the same length as that used for adults. The tip of the tube impinged upon the carina and caused considerable coughing. It also interfered with the passage of air into the mainstem bronchi and resulted in cyanosis. When the tube was shortened, the situation was rectified.

Instances have occurred in which the tracheotomy tube was obstructed by secretions and resulted in interference with respiratory exchange. If too vigorous suction was exerted by the nurses or doctors in attendance, it caused added inflammatory edema of the tracheobronchial tree and bleeding from the eroded mucosa surface, and further increased the amount of secretions that had to be cleared.

Patients have been received with the tracheotomy opening made as a linear slit, often on the side of the trachea. This always results in poor exchange, owing to the necessity of using a small tube, erosion because of improper fit, and extreme difficulty with anesthesia. The only satisfactory technique is to remove a block of cartilage in the anterior tracheal wall.

Summary

Experience with the use of tracheotomy in burns is reviewed. As a prophylactic procedure it definitely facilitates care, but its full value is difficult to determine in this small series. The therapeutic use of tracheotomy appears of little value except in emergency instances of acute localized obstruction.

If tracheotomy is performed adequately and the patient cared for properly, complications should be minimal.

Suite 208, Sheffield Building, 1938 Peachtree Rd., N. W. (9).

Subdural Hematoma Following Spinal Anesthesia

KEASLEY WELCH, M.D., Denver

Of the recognized neurological sequelae of spinal anesthesia, those due to lumbar puncture are generally benign but relatively common, while those due to the constituents of the injection are, as a rule, serious but infrequent. To the list of hazards must be added a complication which, though due to lumbar puncture, is grave but very rare. The following case reports illustrate the occurrence of subdural hematoma as a consequence of spinal anesthesia.

Report of Cases

CASE 1.—The patient was a 69-year-old retired chiropractor who had suffered from chronic sinusitis and bronchiectasis for many years. After an episode of urinary retention, he underwent, on May 23, 1954, a retropubic prostatectomy and vasectomy under spinal anesthesia. The lumbar puncture was made between L-2 and L-3 with a No. 22 needle. Tetracaine hydrochloride, 14 mg., and epinephrine, 0.4 mg., were administered. During the operation he coughed and expectorated several times. Although his recovery was otherwise satisfactory, he developed, during the postoperative period, bilateral frontal headache, which he blamed upon the spinal anesthesia. On July 7, 1954, he complained of diplopia, and on the following day he became very drowsy and confused. These symptoms persisted through July 9, and on that night he fell from his bed. On July 10, he ate breakfast, but afterward became drowsier and more difficult to arouse, and was then admitted to Colorado General Hospital.

On physical examination, he was found to be emaciated and uncooperative. He would neither answer questions nor obey commands for one examiner, but a little later, when seen by another, was awake and fairly cooperative but disoriented. Blood pressure was 120/80; temperature 37 C (98.6 F); pulse 80 a minute.

The skin was of poor turgor and hydration, and his lips were dry. The remainder of the general examination was normal except for deviation of the trachea to the left and the presence of an apical systolic murmur.

The neurological examination, limited as it was by the patient's ability to cooperate, disclosed the varying state of consciousness previously mentioned. The cranial nerves functioned within normal limits except for the complaint of diplopia. Movements were well performed with either arm or leg. The tendon reflexes were symmetrical. The plantar response, initially flexor, was found later to be extensor on both sides. The superficial abdominal reflexes could not be elicited.

Operation.—On July 11, 1954, bilateral burr holes were made. Dark subdural blood was found bilaterally and drained.

Course.—There was improvement in his state of consciousness for three days, after which he again became very drowsy. Aspiration of 30 ml. of fluid through the burr holes restored his alert state for a day, but drowsiness, progressing to coma, began again on the fifth day and was not altered by reexploration of the subdural space on the sixth day. He died July 19.

The autopsy findings included bilateral bronchopneumonia, multiple infarcts of the brain, and the membranes of the subdural hematomas, containing small amounts of fluid.

A retropubic prostatectomy under spinal anesthesia was followed, in this 69-year-old man, by headache and, later, drowsiness, confusion, and stupor. Bilateral subdural hematomas were found and drained; but the patient's improvement was transitory, and he died on the eighth postoperative day.

CASE 2.—The patient, a 42-year-old woman, was admitted to Denver General Hospital on May 23, 1957. On Dec. 21, 1956, she had had a cholecystectomy at another hospital under continuous spinal anesthesia, procaine hydrochloride, 175 mg., being used. She experienced hiccups during the operation. From the time of her operation, she had suffered from left-sided headache, which was severe and persistent. For two days prior to admission, she had been nauseated and had vomited several times. On the day of admission, she had a series of seizures.

Physical Examination.—At 5- to 10-minute intervals the patient was having repeated seizures, beginning with clonic movements in the right arm

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From the Division of Neurosurgery of the University of Colorado School of Medicine.

and spreading to the right leg and right side of the face, together with head and eye deviation to the right. Immediately after an attack the tendon reflexes on the right were more active than those on the left and there was right ankle clonus, but within minutes the reflexes were again symmetrical. There persisted, however, mild weakness of the right upper extremity. The remainder of the examination failed to reveal abnormalities.

After the attacks were controlled, plain x-rays of the skull were made, and a calcified pineal was seen to be displaced from the midline toward the right.

Angiography.—A left carotid angiogram was made. The anterior cerebral artery was displaced to the right, and there was displacement of the surface branches of the middle cerebral artery away from the skull.

Operation.—On the evening of admission a left parietal burr hole was made. Upon opening the dura, a green membrane was disclosed, and beneath this a watery subdural hematoma of moderate size. This was drained.

Course.—On the second postoperative day the patient became restless and confused, experienced visual and auditory hallucinations, and expressed delusions that members of the staff were trying to kill her. On the following day she was again coherent and well oriented, and recovery was uninterrupted afterward. She was discharged home June 3, 1957, and on July 1, 1957, neurological examination was normal, although she still complained of some headache.

After a cholecystectomy under continuous spinal anesthesia, this 42-year-old woman complained of persistent and severe left-sided headache. She was admitted to the hospital five months later, having repeated right-sided seizures. A left subdural hematoma was drained the same day, and recovery occurred.

Comment

Rarely, the release of cerebrospinal fluid may so reduce intracranial pressure and alter stability of the intracranial contents that subdural bleeding occurs. In those situations where this mechanism has been recognized as leading to subdural hemorrhage, the removal of spinal fluid has usually been both rapid and gross. Thus, Smith and Crothers [8] described subdural effusion following pneumoencephalography in infants. They ascribed the effusion to the tearing of the veins passing from the cortex to the dural sinuses consequent to the removal of support of the spinal fluid. Similarly, Anderson [1] and Davidoff and Feiring [5] described subdural hemorrhage following operations involving the release of cerebrospinal fluid in children with hydrocephalus. In adults, subdural hematoma has followed pneumoencephalography,[3,7] the surgical removal of spinal tumor,[4] and, in a personally observed case, the opening of the theca for chordotomy.

In 1949 Gerlach [6] described the case of a 66-year-old man who developed persistent headache after spinal anesthesia. Ptosis and progressive stupor ensued. A subdural hematoma was evacuated on the 10th day. He discussed the relationship of reduced intracranial pressure to the formation of the hematoma. This is the only recognized and treated case of subdural hematoma ascribable to spinal anesthesia which I have found. Bodechtel,[2] however, described at autopsy a large subdural hematoma associated with small foci of intracerebral hemorrhage following the administration of spinal anesthetic.

In reviewing our records, we have found other instances of subdural hematoma in patients who have had spinal anesthesia where no relationship could be shown to exist—instances, for example, where an adequate history of head trauma preceded the onset of symptoms. In the cases presented here, there was no history of head injury, and in each, symptoms began with the spinal anesthesia and were ascribed by the patient to the spinal anesthesia.

The majority of reviews devoted to a consideration of complications of spinal anesthesia do not mention subdural bleeding, an exception being that of Thorsen,[9] who cited Bodechtel's case. His analysis of published cases in which delayed cerebral effects occurred after spinal anesthesia indicates that venous-sinus thrombosis was often responsible, while multiple petechial hemorrhages in the brain, cerebral infarction, and subarachnoid hemorrhage also occurred.

The diagnosis of subdural hematoma ought to be considered in patients with per-

sistent post-spinal-anesthesia headache and in those who develop confusion, dementia, and somnolence, with or without motor disturbance after spinal anesthesia.

Summary

Attention is directed to the rare occurrence of subdural hematoma as a complication of spinal anesthesia, and two cases illustrating this sequence are presented.

Division of Neurosurgery, University of Colorado School of Medicine, 4200 E. 9th Ave. (20).

REFERENCES

1. Anderson, F. M.: Subdural Hematoma, a Complication of Operation for Hydrocephalus, Pediatrics 10:11-17 (July) 1952.

2. Bodechtel, G.: Befunde am Zentralnervensystem bei Spätnarkosetodesfällen und bei Todesfällen nach Lumbalanästhesie, Ztschr. ges. Neurol. u. Psychiat. 117:366-423, 1928.

3. Bucy, P. C.: Subdural Hematoma, Illinois M. J. 82:300-310 (Oct.) 1942.

4. Cooney, J. F., and Baker, G. S.: Subdural Hematoma Following an Operation on the Spinal Cord: Report of a Case, Proc. Staff Meet. Mayo Clin. 28:364-367 (July 1) 1953.

5. Davidoff, L. M., and Feiring, E. H.: Subdural Hematoma Occurring in Surgically Treated Hydrocephalic Children with a Note on a Method of Handling Persistent Accumulations, J. Neurosurg. 10:557-563 (Nov.) 1953.

6. Gerlach, J.: Subduralhämatom und erniedrigter Schädelinnendruck, Deutsche Ztschr. Nervenh. 160:387-399, 1949.

7. Robinson, R. G.: Subdural Haematoma in an Adult After Air Encephalography, J. Neurol. Neurosurg. & Psychiat. 20:131-132 (May) 1957.

8. Smith, H. V., and Crothers, B.: Subdural Fluid as a Consequence of Pneumoencephalography, Pediatrics 5:375-389 (March) 1950.

9. Thorsen, G.: Neurological Complications After Spinal Anaesthesia and Results from 2,493 Follow-Up Cases, Acta chir. scandinav. (Supp. 121) 95:1-272, 1947.

Some Problems Encountered in Treatment of Peripheral Arterial Disease

THOMAS G. BAFFES, M.D.; CLARENCE NORBERG, M.D., and SPYROS KATOPODIS, M.D., Chicago

During the past three years, we have had the opportunity to treat some 75 patients for peripheral arterial disease. In their management, currently accepted methods for translumbar aortography and femoral arteriography [1,2] were utilized to supplement clinical diagnosis, and the usual methods for thromboendarterectomy,[3] lumbar sympathectomy,[4] and introduction of arterial grafts [5-8] were applied. However, along with the good results that followed these methods, we have had some sobering experiences, emphasizing that success depends not only on proper diagnosis and surgical techniques but also on a number of extraneous factors that sometimes cannot be accurately delineated. This article emphasizes some of these factors. Most of them are hemodynamic. For the most part, the role they might play in a specific instance is difficult to predict before operation. Yet the possibility of their existence must be constantly kept in mind in evaluating patients with peripheral vascular disease.

Hematometakinesis

Hematometakinesis is defined as the shifting of blood flow from one section of the body to another.[9,10] This usually occurs in response to differences in pressure resulting from regional changes in the capacity of the vascular bed. Specifically, in peripheral arterial disease, this phenomenon occurs when one section of the patient's cardiovascular system responds extremely well to a given method of treatment, while another section of the cardiovascular system responds poorly or is not adequately included in the plan of treatment. That section of the cardiovascular system which responds well may drain off the major portion of the available circulation, leaving the section that responds poorly relatively ischemic. The results that follow in the ischemic areas may be temporary or permanent and may vary from merely an increase in symptoms to frank gangrene. In our series of patients there were three such instances.

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From the Department of Surgery, Northwestern University School of Medicine, and from the Augustana Hospital, Swedish Covenant Hospital, and Children's Memorial Hospital.

Report of Cases

CASE 1.—A 59-year-old man was admitted to the hospital complaining of pain in both lower extremities and in his back. Pain in the right extremity was localized in the hip and thigh. There was also pain in the right toes suggestive of peripheral arteritis. In the left lower extremity, pain was primarily in the calf and ankle. On being questioned regarding intermittent claudication, the patient related the pain in his limbs to exercise, but felt that pain in the left lower extremity was aggravated by exercise much more than the pain on the right. He stated that he had to stop walking primarily because of pain in the left calf. He had also noted "rest pain" at night on the left side.

Pulsations of both the right and left femoral arteries could be felt. Pulsations of the popliteal, dorsalis pedis, and posterior tibial arteries were present on the right side but absent on the left. Roentgenograms showed hypertrophic arthritis in the lumbar vertebrae. Arteriograms revealed segmental occlusion of the left superficial femoral artery, with multiple atheromatous plaques in the left popliteal artery and its branches that made a "by-pass" operation impossible. The major arterial system of the right lower extremity did not have any significant obstructions, though a few atheromatous plaques were seen (Fig. 1). It was decided to perform bilateral lumbar sympathectomy.

Following operation, there was marked increase in temperature on the right side. There was very

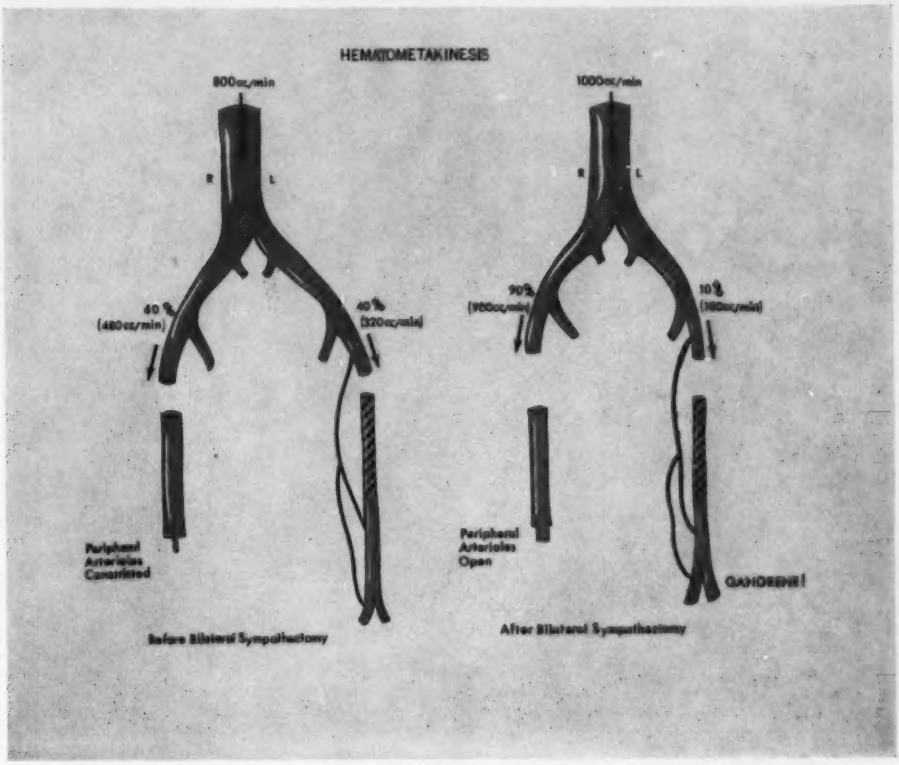

Fig. 1.—Diagram illustrating shift of blood flow from the left lower extremity to the right. This occurred after marked dilatation of peripheral arterioles on the right with relatively poor dilatation on the left. The flow rates shown are arbitrary estimates and are used merely for illustration.

poor temperature response on the left. The left foot and calf remained cold, and pain became continuous and severer. During the ensuing weeks, there was no improvement in symptoms. A few months later, gangrene developed in the left first and second toes.

In retrospect, it was apparent that most of the symptoms on the right were due to hypertrophic arthritis. Arterial involvement there was relatively mild. After bilateral lumbar sympathectomy, there was a massive response on the right with little or no vasodilatation on the left. A great deal of the total available circulation was shifted from the left to the right extremity, leaving the left extremity more ischemic than before operation.

CASE 2.—A 65-year-old woman was admitted to the hospital with a chronic ulcer on the anterior aspect of her left leg, and continuous rest pain in that region for four months before admission. The rest pain was relieved by hanging the leg in the dependent position. For about one year before admission, she had suffered from intermittent claudication in the involved limb. Recently, it had become dusky and colder.

There were no pulsations in the left femoral, popliteal, posterior tibial and dorsalis pedis arteries. Roentgenograms before operation revealed calcification in the iliac, femoral, and popliteal vessels. Operative arteriograms showed segmental occlusion of the iliac, common femoral, and superficial femoral arteries, with collateral vessels in the pelvis and thigh, but little collateral circulation at the knee (Fig. 2). The "run-off" could not be visualized, and, at operation, it was obviously inadequate. The only therapeutic procedure that could be done was left lumbar sympathectomy. Following operation, the patient experienced temporary relief of rest pain. However, the temperature of the left leg and foot did not rise, and there was no improvement in color. By the third postoperative day, it was apparent that ischemia of the left leg and foot was increasing, although the skin temperature of the thigh had improved. Gangrene of the toes developed and amputation had to be performed.

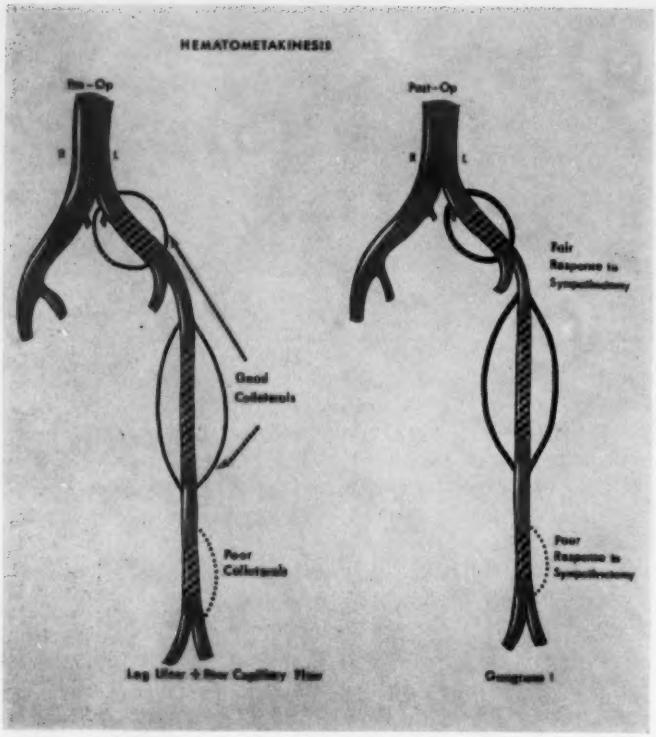

Fig. 2. — Diagram of operative arteriograms in Case 2. Response to left lumbar sympathectomy in the thigh and pelvis was fair. Response in the leg was poor. Gangrene was precipitated in the left toes.

CASE 3.—A 57-year-old male alcoholic was admitted to the hospital with a history of bilateral intermittent claudication in his lower extremities for six months before admission. There were multiple chronic ulcers on both extremities suggestive of avitaminosis and ischemia. These had failed to respond to antibiotics, dressings, and vitamin therapy. On physical examination, there was absence of the left femoral, popliteal, dorsalis pedis, and posterior tibial arteries. The right femoral artery could be palpated, although its pulsations were decreased. The right popliteal, dorsalis pedis, and posterior tibial arteries could not be felt. Aortography and bilateral femoral arteriography revealed segmental occlusion in the left iliac artery with scattered plaques in the right femoral artery (Fig. 3).

Because the symptoms were severest on the left side, the segmental occlusion of the left iliac artery was operated on first. A Teflon prosthesis was used to by-pass the occluded iliac artery. Left lumbar sympathectomy was also performed in order to provide a good "run-off" for the Teflon graft. The immediate postoperative result was excellent. The left popliteal and posterior tibial arteries could be readily palpated, and the temperature of the left foot and leg was greatly increased. The chronic skin lesions improved in color and began to heal. However, circulation of the right lower extremity was markedly decreased. The right leg became colder, and the patient began to complain of increased pain in the right calf. About 10 days after operation, there was extensive thrombosis in the right superficial femoral artery, which did not respond to lumbar sympathetic blocks. Gangrene developed in the right foot, and amputation had to be done.

Comment.—In all three cases, therapy was administered which had proved useful on many other similar occasions. Yet, instead of the expected improvement in the ischemic limbs, such shifting of the circulation occurred in response to differences in vasodilatation that the ischemia was aggravated. In evaluating each case to find some means of predicting the unhappy event, we found nothing unusual. The patients represented complicated cases of peripheral arterial disease; yet these often respond well to treatment. The only possible warning sign might have been the history of rest

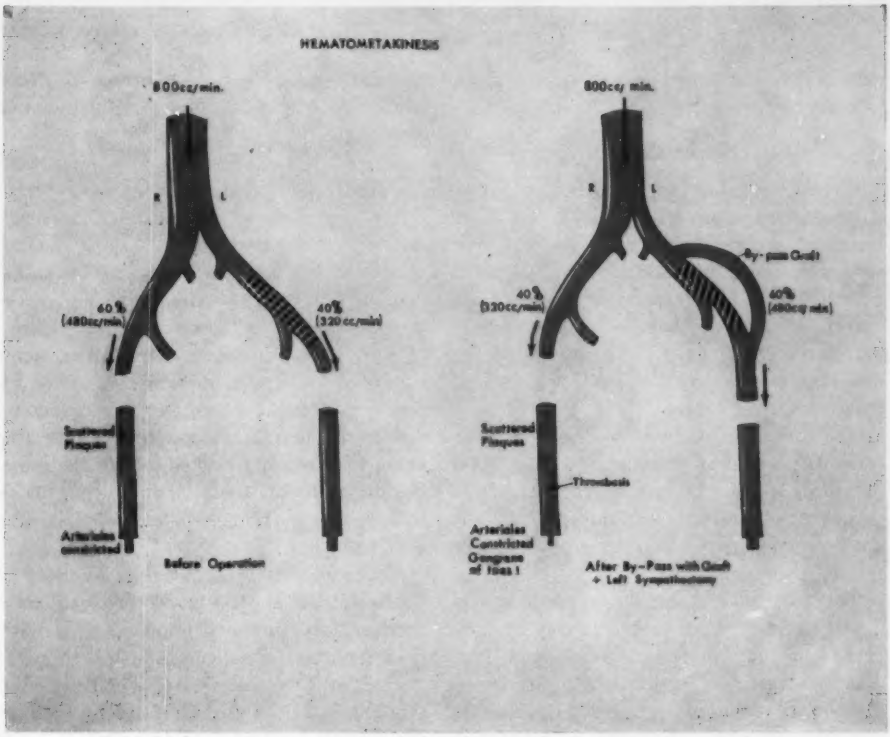

Fig. 3.—Diagram illustrating hematometakinesis resulting from by-pass graft and sympathectomy on the left. The figures are only estimates but they demonstrate how this phenomenon reduced blood flow on the right side enough to permit acute thrombosis of the right superficial femoral artery.

Fig. 4.—Arteriogram showing plaque in the distal portion of the superficial femoral artery which partially occludes the vessel yet permits enough blood flow to make the limb relatively asymptomatic. These "silent" plaques are the source of acute thrombosis following hypovolemia.

pain which was noted in two of the three patients.

Hypovolemia

Hypovolemia has long been recognized as an important cause of coronary occlusion. Surgeons are keenly aware of the necessity for maintaining the patient's blood pressure during operations, especially in cases of known coronary ischemia. A number of "cardiac arrests" have been traced to coronary occlusion secondary to hypovolemia. Internists, too, know that patients with acute coronary occlusion should receive treatment aimed at maintaining their blood pressure, lest the occlusion progress. It is surprising, therefore, that this process has not been emphasized in other types of acute arterial thrombosis.

The particular cause for hypovolemia is not of primary significance. It may be dangerous in *any* arteriosclerotic vessel in which it occurs. It is enough for blood flow to be reduced in a vessel which is partially occluded (Fig. 4). Thrombosis can occur distal to the occlusion, and, depending on the organ involved, may complicate the patient's previous condition to such an extent that the outcome is disastrous. This was dramatically illustrated in four patients.

One patient, a 65-year-old woman, was involved in an automobile accident and sustained contusion of the chest wall. On admission to the hospital, she complained of abdominal pain far in excess of the initial trauma. During the next six hours, she developed signs of peritonitis and laparotomy was performed. At operation, acute thrombosis of the superior mesenteric artery was found, necessitating extensive resection of the jejunum, ileum, and ascending colon. She ultimately died, not of the initial trauma, but of acute arterial thrombosis that resulted from hypotension following that trauma.

Three patients, after being admitted to the hospital in shock—in two instances from coronary occlusion and in one from cerebral hemorrhage—developed acute thrombosis of a superficial femoral artery. Their convalescence was seriously complicated by the sudden appearance of a cold, pulseless lower extremity that required immediate thromboendarterectomy. In two of the patients, thromboendarterectomy was done early

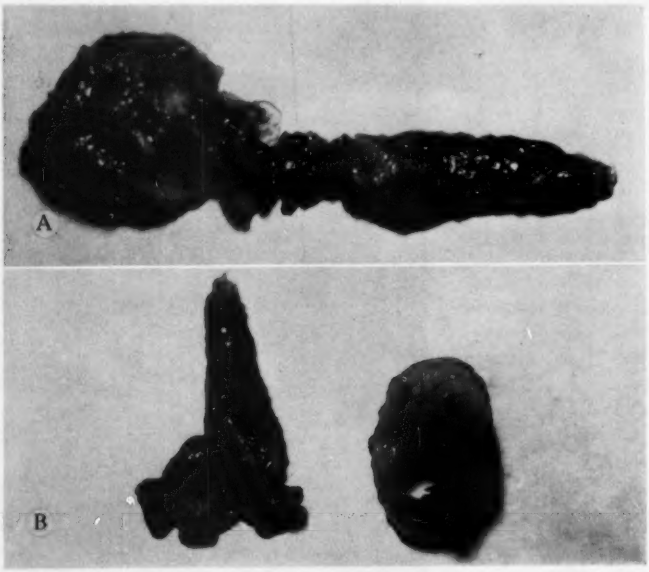

Fig. 5.—*A*, photograph of plaque and distal thrombus which were removed from a superficial femoral artery such as the one described in Figure 4. *B*, note that the thrombus had extended down to the bifurcation of the popliteal artery. The plaque surrounded the artery, yet had a lumen in its center.

Fig. 6.—Photograph of relatively ischemic leg with dry, mummified gangrene of three toes. These were treated conservatively until demarcation and spontaneous amputation occurred. The patient still walks on his own limb.

enough, and circulation was successfully restored (Fig. 5). In the other patient, thrombosis recurred following operation.

Local Trauma

It has long been recognized that local trauma may initiate gangrene in the arteriosclerotic lower extremity. For the most part, local trauma is supplied by the patient himself and takes the form of actual physical damage to the limb, infection or ill-advised home surgery on ingrown toenails, soaking of the feet in hot solutions, application of caustic proprietary medicines, wearing tight shoes, and others. The physician has little control over these events, except to promote good public education in order to prevent them. But what of the local trauma that is supplied by the over-enthusiastic doctor himself?

When gangrene is encountered in an arteriosclerotic limb, the first impulse is to amputate the offending part. There can be no quarrel with amputation in the presence of infected, wet gangrene. However, when the gangrenous area is dry, mummified, and free of infection, the surgeon should consider the possibility that he may well preserve the patient's limb for a longer time if he does not remove the area of gangrene. The mummified part, especially when protected by antibiotics, acts as an excellent dressing, and in time will be amputated spontaneously. If the surgeon avoids the local edema that inevitably follows amputation and concentrates on improving the circulation to the rest of the extremity (e. g., by sympathectomy or arterial graft), the ischemic limb may be usable. Any painless limb,[11] even one lacking a few digits, is better than midthigh amputation and a prosthesis (Fig. 6).

Six patients in our series were treated by judicious neglect of dry, mummified

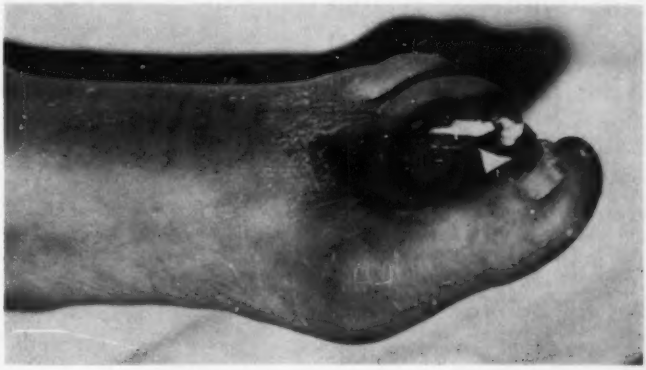

Fig. 7. — Photograph showing a rather frequent result of surgical amputation of dry gangrenous toes. Following amputation of the second toe, edema in the adjacent tissues resulted in spread of the gangrene. This denuded and gangrenous area then became infected. Ultimately, the entire leg had to be amputated.

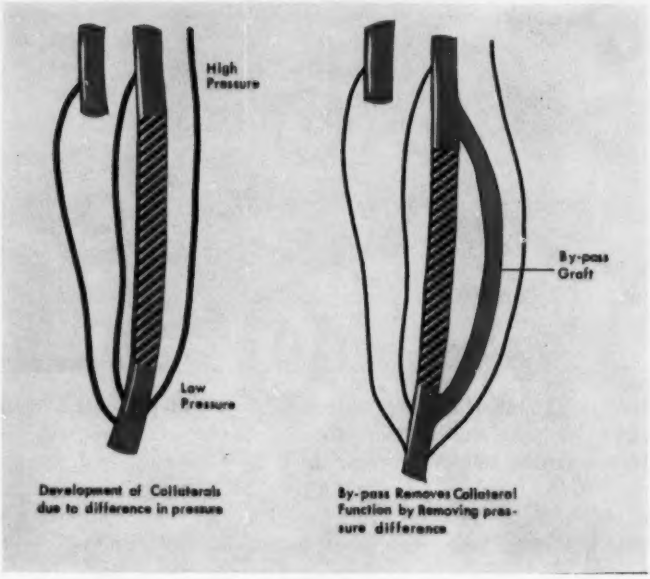
High Pressure
Low Pressure
By-pass Graft
Development of Collaterals due to difference in pressure
By-pass Removes Collateral Function by Removing pressure difference

Fig. 8.—Diagram illustrating decrease in collaterals about an obstruction, following introduction of by-pass graft. This results because greater blood flow through the graft reduces pressure above the point of obstruction (a type of local hematometakinesis). If the graft subsequently clots, gangrene may result because the protective collaterals cannot re-open in time.

digits. The longest follow-up has been for three years. In all six, the mummified areas are still intact or have been amputated spontaneously. None of these patients are amputees. In two other patients, amputation of a mummified digit led to local edema and spread of gangrene. Since the skin had been broken, the new gangrenous areas inevitably became infected and ultimately led to loss of the ischemic limb (Fig. 7).

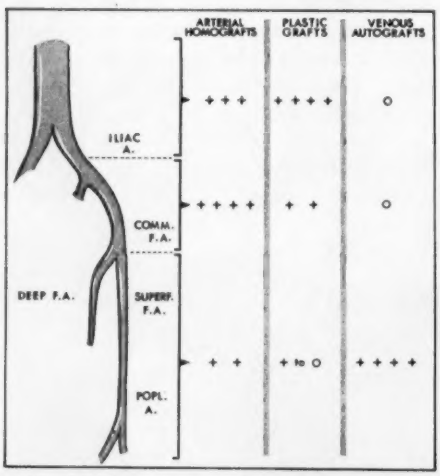

Fig. 9.—Diagram illustrating our current preference for plastic, arterial, or venous prostheses in the lower aorta, its tributaries, and the vessels to the lower limbs. Preference is determined by availability, condition of the peripheral circulation, and expected rate of successful implantation.

Peripheral and Collateral Circulation

The condition of the peripheral and collateral circulation is of utmost importance in determining the type of therapy that is administered to a given patient with peripheral arterial disease. All too frequently, the surgeon finds himself risking the ultimate viability of the patient's ischemic limb upon success of the therapeutic procedure he performs (Fig. 8). This is especially true of arterial implants. For this reason, we have become more and more cautious in the introduction of plastic arterial prostheses in smaller vessels, where our laboratory work indicates that they do not have as high an incidence of successful anastomoses as do homologous arterial or autologous venous implants. As shown in Figure 9, plastic arterial substitutes are introduced freely in the abdominal aorta and the common iliac vessels. However, in the common and super-

ficial femoral arteries, only those situations which have completely patent "run-offs" and which require relatively short segments are considered suitable for plastic implants. If there is any doubt concerning the peripheral vascular bed, an autologous venous prosthesis is used for the superficial femoral artery. Venous prostheses have been eminently satisfactory when introduced in the adductor canal, where external muscular support is available. There, excessive dilatation and formation of aneurysms in venous grafts has not been a problem. Where there is no external muscular support (e. g., in the common femoral artery), homologous arterial grafts are used. These venous and arterial grafts have succeeded consistently despite atheromatous plaques and sluggish flow in the "run-off." It is recognized that this policy is not entirely in keeping with the current enthusiasm for plastic implants. However, we feel that we have salvaged a greater number of lower limbs by this conservative approach. Our patients, unfortunately, have often had such complicated forms of peripheral arteriosclerosis that only about 10% have been suitable for introduction of plastic grafts.

In addition, we have always made liberal use of lumbar sympathectomy wherever the peripheral or collateral circulation of the ischemic limb might be improved by this procedure. It has been particularly applied where arterial prostheses could not possibly be introduced. There is no doubt that it has often relieved symptoms and saved limbs.

When lumbar sympathectomy is used, the physician should appreciate the fact that its results are not as dramatic as those of successful arterial implants. It is not intended to compete with the latter, only to supplement it. Sympathectomy facilitates dilatation of vessels—to the extent that the arteriosclerosis present permits that dilatation to occur. It does nothing more. In severe disease, several weeks may be required for improvement. However, when the limitations of the procedure are recognized, it can be applied with good effect not only in desperate cases of peripheral arteriosclerosis but also to improve the results of arterial grafts.

Associated Disease

It is not unusual, when dealing with older patients, afflicted with peripheral arterial disease, to find multiple types of disorders. Unless the surgeon is constantly alert for this situation, he may attempt to correct arterial disease in the lower extremities in a patient who has an unrelated lesion which may contraindicate any surgery at all. Thus, in our experience, two patients were presented for treatment of intermittent claudication who had inoperable pulmonary carcinomas, and one patient had laparotomy for proved abdominal aneurysm, only to encounter a silent inoperable gastric carcinoma. Sometimes, arteriosclerotic disease itself may be so extensive as to make surgery difficult or impossible. We have often had to care for intermittent claudication in patients who had histories of previous coronary occlusion, cerebral vascular accidents, angina, or hypertension. These hazards are familiar to all, and are mentioned only to reemphasize the need for thoroughly evaluating the patient before embarking upon treatment of his peripheral arterial disease.

Keeping in mind the possibility of multiple lesions, however, is of great value not only because it helps avoid hopeless cases or unnecessary complications, but also because the coexisting multiple lesions may *all* require treatment before the patient's symptoms are completely relieved. Thus, a patient may have severe varicose veins in conjunction with arterial obstruction, each condition aggravating the chronic ulcers on his leg (Fig. 10). Another patient with intermittent claudication secondary to arteriosclerosis may have an associated neuropathy which is causing foot drop. The patient's symptoms cannot be completely alleviated by correcting the ischemia of his limb alone. He also needs some type of supporting brace before he can be completely ambulated (Fig. 11). Arthritis commonly

Fig. 10.—Patient having combined arteriosclerosis of superficial femoral and popliteal arteries, and varicose veins. He was treated by left lumbar sympathectomy followed by stripping of the varicose veins. The ulcers subsequently healed.

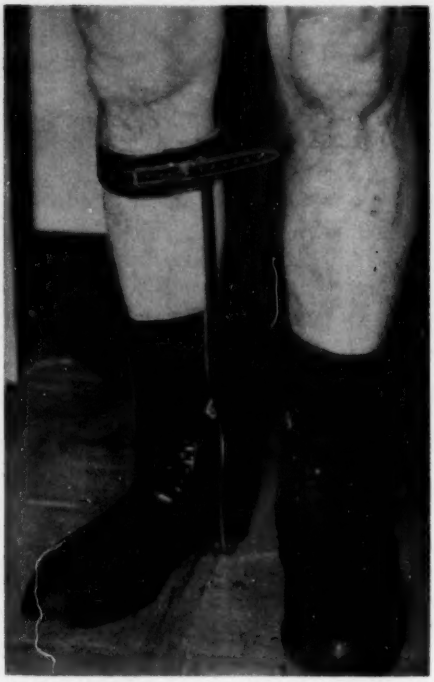

Fig. 11.—Patient having bilateral intermittent claudication in addition to right foot drop. Treatment involved bilateral lumbar sympathectomy plus application of lower leg brace.

masks or aggravates the symptoms of arteriosclerosis (e. g., Case 1). These are common examples in the experience of any practicing physician. The fact remains that complete alleviation of the patient's difficulty may require not only correction of ischemia but also treatment of other coexisting lesions as well.

Problems in Children

Peripheral arterial disease occurs in children, but it is unique. It is seldom associated with arteriosclerosis, although a 3-day-old infant dying of acute coronary occlusion has been seen in the Children's Memorial Hospital. Usually, peripheral arterial problems in children have to do with structural deformities, neoplasms, or congenital abnormalities of the vessels themselves.

There were two instances in our experience, in which the abdominal aorta was involved by neoplasm and had to be sacrificed. In one infant, the abdominal aorta and its bifurcation was replaced with a homologous graft. However, the lumen of the iliac vessels was only about 2 mm. in diameter, and the homograft soon clotted. In the other child, the abdominal aorta was excised along with an invading neuroblastoma and was not replaced. In both instances, the circulation of the lower extremities, although moderately impaired in the immediate postoperative period, remained adequate to maintain viability of the lower extremities. Within a few weeks after operation, collateral circulation had become well established and the lower extremities were warm. The patients have now been followed for two years. Femoral artery pulsations are now palpable. Their

walking has not differed, in development or ability, from that of other children of their age.

Two children were operated upon for aneurysms, one in the popliteal and the other in the axillary artery. The popliteal aneurysm was saccular and could be excised without the introduction of an arterial graft. In the other child, aged 10 years, the aneurysm of the axillary artery was excised and replaced with a homologous arterial graft. The patient has been followed for two years, and the graft has remained patent.

Two children were treated for solitary, congenital arteriovenous fistula associated with hemangioma formation. One had a fistula between the left posterior tibial artery and vein, but the arteriovenous communications were so diffuse that exploration failed to reveal a local lesion that could be excised. All the potential tributary arteries were dissected out and ligated. However, within a few months, collateral circulation had developed, and the arteriovenous hum could be heard once again. The other patient had a similar history, except that the diffuse arteriovenous fistula was associated with hemangioma of the scrotum and thigh (Fig. 12). In this patient, the common iliac, common femoral, and superficial and deep femoral arteries were dissected free, and all potential branches to the hemangioma were ligated. The multiple ligations even included the deep femoral and the hypogastric arteries. Yet, within six weeks, the murmur and pulsations were again noted in the hemangioma.

Fig. 12.—Roentgenogram showing extensive congenital arteriovenous fistula associated with hemangioma of scrotum and thigh.

Summary

A review of factors affecting our results in the surgical treatment of 75 patients with peripheral arterial disease is presented.

Each factor is discussed and specific examples are presented for illustration.

It is emphasized that many of these factors are independent of surgical technique and aortography or arteriography. Examples are used to demonstrate how their influence may often be very difficult to predict before operation.

Peripheral arterial problems encountered in children are described and are compared with those encountered in adults.

4055 Main St. Skokie, Ill. (Dr. Baffes).

REFERENCES

1. Szilagyi, D. E.: Lumbar and Peripheral Arteriography, Radiology 69:177, 1957.

2. Menendez, C. V., and Linton, R. R.: A Technique of Abdominal Aortography and Femoral Arteriography, Surgery 40:718, 1956.

3. Muller, W. H., Jr.; Liddle, H. V., and Edmunds, M. C., Jr.: Surgical Management of Arterial Obliterative Disease: Special Reference to Thromboendarterectomy, Ann. Surg. 147:845, 1958.

4. De Bakey, M. E.; Creech, O., and Woodhall, J. P.: Evaluation of Sympathectomy in Arteriosclerotic Peripheral Vascular Disease, J. A. M. A. 144:1227, 1950.

5. Moore, T. C., and Kajikuri, H.: Freeze-Dried and Alcohol Preserved Homografts for Replacement of Small Arteries, Surg. Gynec. & Obst. 103: 155, 1956.

6. Edwards, W. S., and Tapp, J. S.: Chemically Treated Nylon Tubes As Arterial Grafts, Surgery 38:61, 1955.

7. Creech, O.; Deterling, R. A.; Edwards, S.; Julian, O. C.; Linton, R. R., and Shumaker, H.: Vascular Prostheses, Report of the Committee for the Study of Vascular Prostheses of the Society for Vascular Surgery, Surgery 41:62, 1957.

8. De Bakey, M. E.; Crawford, E. S.; Cooley, D. E., and Morris, G. C., Jr.: Surgical Considerations of Occlusive Disease of the Abdominal Aorta and Iliac and Femoral Arteries: Analysis of 803 Cases, Ann. Surg. 148:306, 1958.

9. Ray, T.; Burch, G. E., and De Bakey, M. E.: The "Borrowing-Lending" Hemodynamic Phenomenon (Hemometakinesia) and Its Therapeutic Application in Peripheral Vascular Disturbances, New Orleans M. & S. J. 100:6, 1947.

10. Dorland's Illustrated Medical Dictionary, Ed. 23, Philadelphia, W. B. Saunders Company, 1957 (see Hematometakinesis).

11. Warren, R.; Crawford, E. S.; Hardy, I. B., and McKittrick, J. B.: The Transmetatarsal Amputation in Arterial Deficiency of the Lower Extremity, Surgery 31:132, 1952.

Use of Pneumoperitoneum in Differential Diagnosis of Paracardiac Masses in Children

DAVID H. BAKER, M.D.; BERNARD MAISEL, M.D., and HENRY P. GOLDBERG, M.D., New York

The presence of a mass in the cardiophrenic angle is almost always an incidental finding on an x-ray of the chest taken for another purpose. Special studies, i. e., laminagraphy and angiocardiography, usually do not reveal the nature of the mass, and exploratory thoracotomy is eventually performed. It has been possible to determine the nature of some of these lesions by the use of pneumoperitoneum.

Procedure

The technique of inducing an artificial pneumoperitoneum has been described by several authors.[1-4] In brief, the procedure we have used is described. The patient is placed in the supine position. The abdomen is prepared with benzal konium (Zepharin) and the left upper quadrant draped. A point midway between the left costal margin and the umbilicus is infiltrated with 2% procaine hydrochloride U. S. P.

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A 19-gauge needle 1½ in. long, attached to a 10 cc. syringe, is introduced into the abdominal wall to a point where the anterior rectus sheath is encountered. A needle guard or clamp is adjusted to the needle to permit safe advancement through the rectus muscle, posterior rectus sheath, and peritoneum. As the needle passes through the peritoneum, the patient may feel slight pain. Five cubic centimeters of procaine hydrochloride is introduced into the peritoneal cavity to test the free flow and to be certain that the needle tip is in the peritoneal cavity rather than in the rectus sheath. A three-way stopcock is attached to the needle, and a small amount of room air is introduced to test the free flow into the peritoneal cavity. The 10 cc. syringe is replaced by a 50 cc. syringe, and about 250 to 350 cc. of air is injected (Fig. 1). The syringe works easier if the barrel is wetted with procaine.

Fig. 1.—Air may safely be introduced into the peritoneal cavity through a 19-gauge, 1½ in. needle, employing a three-way stopcock and a 50 cc. syringe. The skin is infiltrated with 1% procaine hydrochloride at a point 3 cm. cephalad to the umbilicus and 2 cm. to the left of the umbilicus. As the needle passes through the peritoneum, the patient may feel slight pain. After entering the peritoneal cavity, 5 cc. of procaine is introduced into the peritoneal cavity to test free flow and to be certain that the needle is in the peritoneal cavity, rather than in the rectus sheath.

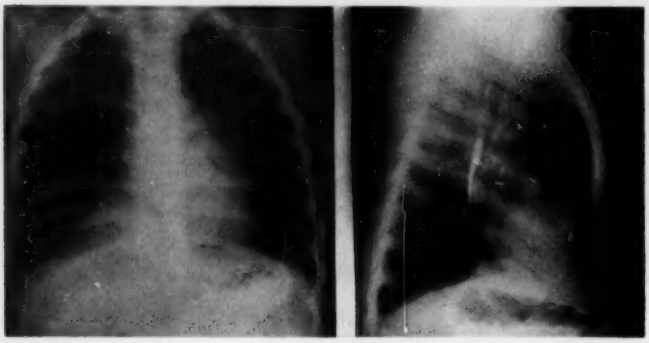


Fig. 2.—X-rays taken prior to induction of pneumoperitoneum revealed a discrete homogeneous mass at the cardiophrenic angle, contiguous with the heart shadow and the diaphragm. Fluoroscopy suggested that it was nonpulsatile and seemed to move with the diaphragm. The diaphragms moved in a normal manner.

After the procedure mild analgesics may be needed to control shoulder pain caused by irritation of air beneath the diaphragm. No complications have been encountered in over 10,000 pneumoperitoneum induced with this technique. However, there have been reported deaths from air embolism.[3,5] These deaths represent a minute percentage when considered in relation to the total number of patients in whom pneumoperitoneum has been induced. In addition, there is general agreement that air embolism is avoidable by strict attention to the details of the technique.[8]

Report of Cases

CASE 1.*—This two-year-old white twin girl was hospitalized for evaluation of an asymptomatic right paracardiac mass discovered on a chest x-ray taken at the age of 18 months because of bronchopneumonia.

A heart murmur, thought to be functional, had been heard since birth. Her general health was good, and development was normal. There were no abnormal respiratory symptoms before or after her bout of bronchopneumonia. The only abnormal finding of the physical examination was a soft apical systolic murmur, without thrills or transmission.

Fluoroscopy revealed a discrete homogeneous mass above the diaphragm, which moved with it, was nonpulsatile, and was located at the right cardiophrenic angle. The diaphragm moved normally (Fig. 2).

No abnormalities of the stomach or small bowel were seen in a gastrointestinal series. The above studies and conventional chest films suggested the presence of a mass above the diaphragm.

Pneumoperitoneum clearly demonstrated herniation of the liver into a medial eventration of the right diaphragm (Fig. 3).

CASE 2.—A 4½-year-old white boy had been followed at this hospital since the age of 4 years because of a seizure disorder and multiple congenital anomalies, including a cardiac malformation. Fluoroscopy for investigation of the congenital heart disease revealed a right cardiophrenic mass. This was less dense than the cardiac shadow, was nonpulsatile, and moved with the diaphragm. The diaphragmatic excursions were normal.

At the age of 4½ years he was hospitalized because of purpura. The physical examination dis-

* Patient referred by Dr. R. Koloski, Poughkeepsie, N. Y.

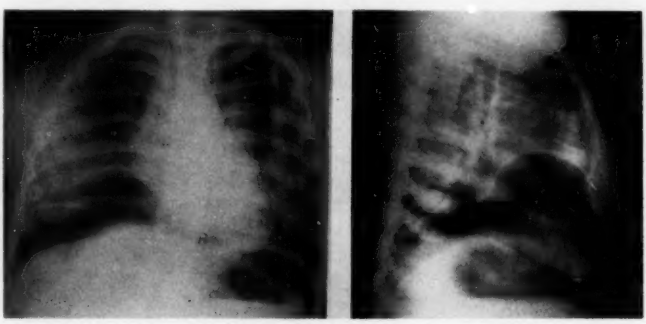


Fig. 3. — Pneumoperitoneum outlined in the x-rays the profile of the diaphragm and clearly indicated that this mass represented a localized eventration of the right diaphragm that was filled with a herniated segment of the right lobe of the liver.

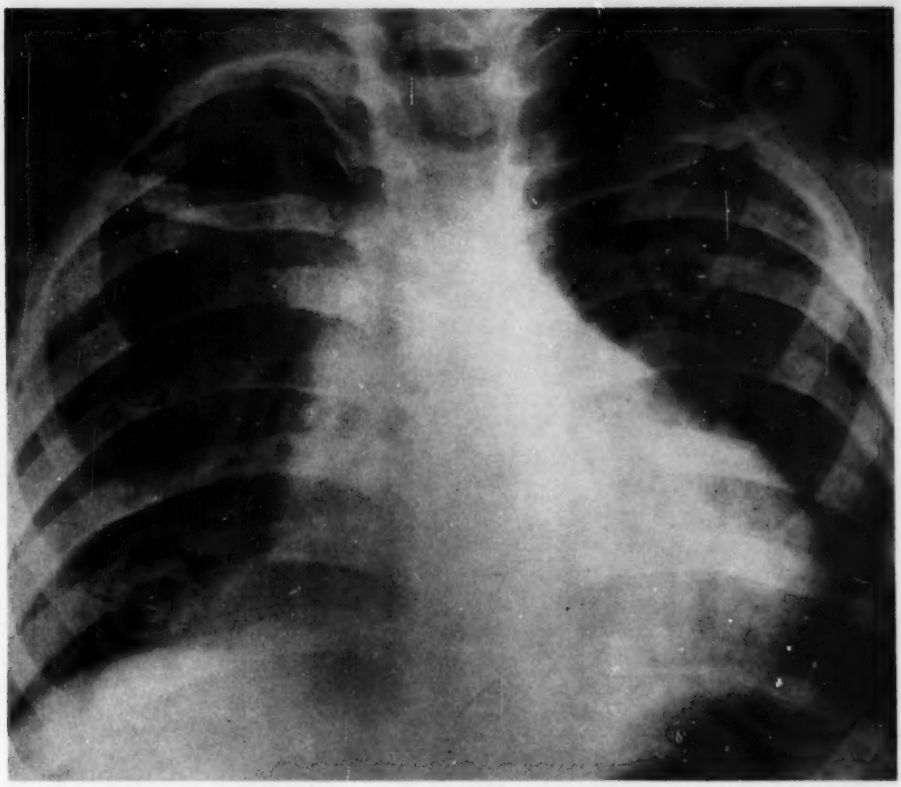


Fig. 4.—An x-ray of the chest disclosed a smooth, oval, homogeneous density at the right cardiophrenic angle, which appeared continuous with the diaphragm and the heart shadow.

closed obvious purpura on the trunk and extremities. There was an organic systolic murmur, believed due to a left-to-right shunt through an atrial septal defect. The lungs were clear to percussion and auscultation.

Laboratory data was unremarkable save for the low platelet count. X-rays of the chest showed a smooth oval density in the right cardiophrenic angle, which appeared continuous with the diaphragm (Fig. 4).

Pneumoperitoneum was induced to confirm the preliminary diagnosis of eventration. Figure 5 shows a small anteromedial eventration of the diaphragm, into which a portion of the liver had herniated.

CASE 3.—This 9-month-old white boy was admitted to the hospital because of fever and anorexia. Fluoroscopy in the outpatient department revealed the presence of a round mass in the right lower lung field, thought to represent inflammatory or neoplastic pulmonary disease.

The physical examination on admission revealed a well-nourished and well-developed male infant, in no distress, who had a diminished percussion note and diminished breath sounds over the right lower anterior chest. The remainder of the examination was unremarkable.

The laboratory work-up showed a normal urine and blood count.

Fluoroscopy revealed a smooth rounded mass just lateral to the right costophrenic angle. It was contiguous with the right diaphragm and moved with the diaphragm. The diaphragmatic excursions were normal. The chest x-ray confirmed the fluoroscopic findings. A barium enema and gastrointestinal series showed no abnormalities.

Pneumoperitoneum was performed and clearly demonstrated a localized eventration of the right diaphragm and herniation of the liver into it. Pneumoperitoneum was repeated after six years and showed no change from the original.

Comment

The portion of the diaphragm involved in an eventration may vary considerably in

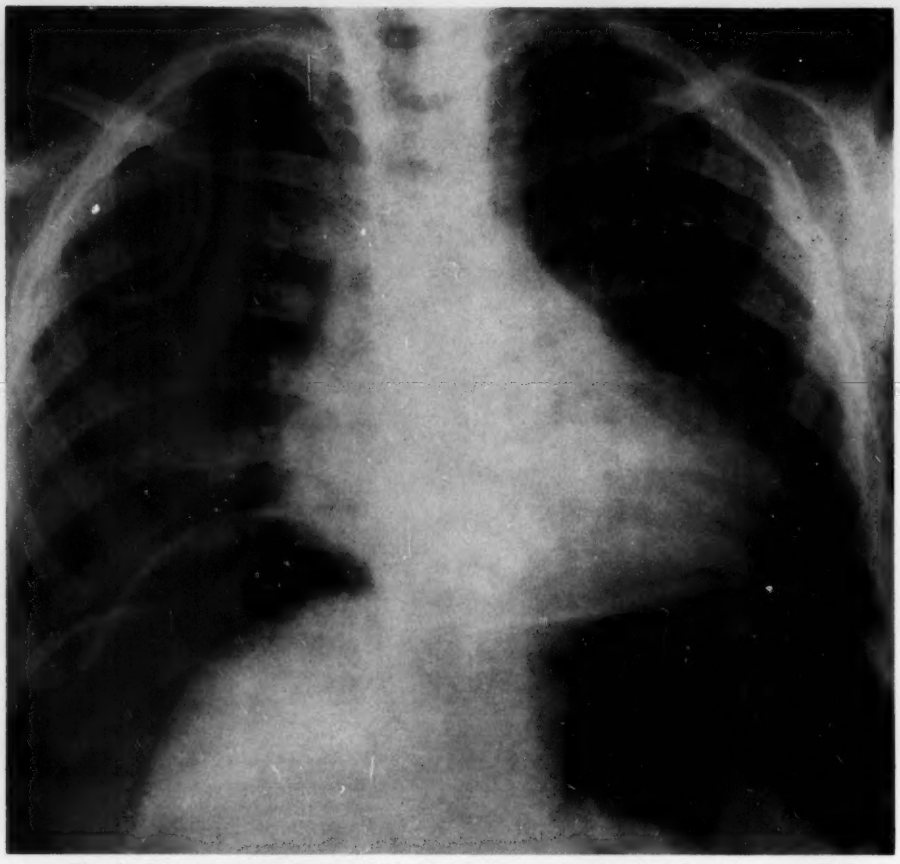


Fig. 5.—Pneumoperitoneum revealed in the x-rays taken with the patient in the upright position the profile of an eventration of the right diaphragm, which was filled with a herniated segment of the right lobe of the liver.

size. Diagnosis of a large eventration is made easily because of the striking symptoms and physical findings, and prompt operative intervention is indicated. Eventration involving only a small portion of the diaphragm may, in a rare instance,[6,7] cause symptoms and require treatment. It was noted from a review of the literature, however, that almost all small eventrations are asymptomatic, discovered incidentally, and compatible with long life. (This includes several cases referred to as accessory lobes of the liver.[8,9]) Clay and Hanlon [10] reported three patients with minor eventrations who had no symptoms when studied at 14, 48, and 70 years of age, respectively, and who were not operated upon after the diagnosis was established by pneumoperitoneum.

Fluoroscopy and x-ray may suggest the possibility of a small eventration but do not exclude other lesions. As seen in Cases 1 and 3, the preliminary studies were misleading. Pneumoperitoneum, on the other hand, provided data for a definitive diagnosis of a minor eventration. Even if an eventration is not present, pneumoperitoneum provides localizing information. In view of the above considerations, it is difficult to justify routine surgical exploration for diagnosis, nor does it appear necessary to correct all such minor defects.[11]

Summary

Pneumoperitoneum is a safe, simple procedure which may yield diagnostic information in small diaphragmatic eventrations, whereas conventional studies may be misleading.

Surgical repair of minor eventrations is usually not necessary.

New York Hospital, 525 E. 68th St. (21).

REFERENCES

1. Maisel, B.; Finby, N., and Evans, J. A.: Pneumoperitoneum As a Diagnostic Aid in the Diaphragmatic Area, Special Exhibit, Mod. Medicine 25:113-124, 1957.

2. Maisel, B.; Cooper, W. C., and Glenn, F.: Pneumoperitoneum in the Managament of Esophageal Hiatus Hernia, J. Am. M. Women's A. 11: 299-315, 1956.

3. Mitchell, R. S.; Hiah, J. S., Jr.; McCain, P. P.; Easom, H. F., and Thomas, C. O.: Pneumoperitoneum in the Treatment of Pulmonary Tuberculosis: Results in 710 Cases from 1937 to 1946, Am. Rev. Tuberc. 55:306-331, 1947.

4. Stein, A., and Stewart, W. H.: Roentgen Examination of the Abdominal Organs Following Oxygen Inflation of the Peritoneal Cavity, Ann. Surg. 70:95-100, 1919.

5. Case, J. T.: A Review of 3 Years' Work and Articles on Pneumoperitoneum, Am. J. Roentgenol. 8:714-721, 1921.

6. Wolfson, S. A., and Goldman, A.: Strangulating Diaphragmatic Hernia of the Liver: Report of Case with Surgical Cure, Surgery 24:846-852, 1948.

7. Bradley, J. E., and Greiner, D. J.: Diaphragmatic Hernia, Am. J. Dis. Child. 66:143-149, 1943.

8. Katz, H. J., and Williams, A. J.: Accessory Lobes of the Liver and Their Significance in Roentgen Diagnosis, Ann. Int. Med. 36:880-888, 1952.

9. Friedman, P. S.; Solis-Cohen, L., and Levine, S.: Accessory Lobe of the Liver and Its Significance in Roentgen Diagnosis, Am. J. Roentgenol. 57:601-603, 1947.

10. Clay, R. C., and Hanlon, C. R.: Pneumoperitoneum in the Differential Diagnosis of Diaphragmatic Hernia, J. Thoracic Surg. 21:57-70, 1951.

11. Ravitch, M. M., and Handelsman, J. C.: Defects in Right Diaphragm of Infants and Children with Herniation of Liver, A. M. A. Arch. Surg. 64:794-802, 1952.

An Amazing Accident: Traumatic Transection of Bronchus and Aorta

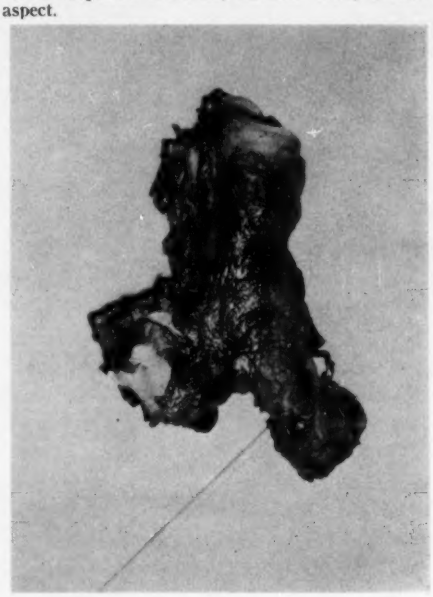
LaMOYNE C. BLEICH, M.D., Ruston, La.

About 10:30 a. m., the morning of Jan. 9, 1958, a 56-year-old white man was brought to the emergency room of the Ruston Hospital. He had been transported in the back seat of a car, sitting erect, by two of his fellow workers. The patient and his friends described that he had been injured by a falling log. The patient was a woodsman engaged in securing pine logs for pulpwood. Apparently, the log had fallen from a truck and hit him across his back. He was completely conscious and talked coherently but complained bitterly of pain in his left chest region. He was having marked respiratory difficulty. A hurried examination, together with a portable x-ray of his chest revealed a complete collapse of his left lung. Despite this, the patient assisted in the examination and cooperated to the extent of rising from the wheel chair and seating himself on the examining table. He was not in shock, having a blood pressure of 128/60, though his pulse was fairly rapid at 100. An intercostal catheter was quickly introduced by means of a trocar into his left anterior chest and connected to an underwater sealed trap. Large quantities of air bubbled forth.

As it was anticipated that this treatment might be inadequate, blood was obtained. The patient was observed for a period of about two hours, and then it was noted that his blood pressure was falling, reaching a level of approximately 60/0.

Blood was started and the patient was taken to the operating room, and a classic left thoracotomy incision was done, and to the amazement of all concerned we noted a complete transection of the left main stem bronchus approximately an inch from the tracheal bification. There was practically no blood in the left chest cavity and only a small ecchymotic area on the anterior aspect of the hilum of the left lung. The left pulmonary artery, which lay immediately over the transected bronchus, was intact and as far as the operators could tell there was no damage to any other structure. The exact mechanism for the transection of this bronchus was obscure for, though he had two broken ribs, they were at a fair distance from the transected bronchus and they were in such good position that they had not been noticed on the x-ray which was taken initially. It was impossible to imagine that this man's chest was sufficiently flexible to have caved in and sprung back without do-

Fig. 1.—Trachea and main bronchi. Pointer indicates repaired bronchus, exterior view, anterior aspect.



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ing further damage to the rib cage. All sorts of conjectures were made: perhaps the bronchus exploded under the force of the blow with a closed glottis, but no conjecture was adequate to explain the injury.

Despite the lack of knowledge as to the mechanism of the injury, the bronchus was repaired with use of interrupted fine cotton sutures, and after its repair the anesthetist was able to inflate the lung quite satisfactorily. The chest was closed, another intercostal catheter first being introduced, and this, together with the one placed preoperatively, was then connected to an underwater trap. Apparently the repaired bronchus was holding nicely, for after the initial air in the chest was expelled very little more issued forth.

During the course of surgery about four units of blood were used, and the patient left the operating room in much better condition than when he entered. Prior to his being returned to his room a bronchoscopic aspiration of the bronchial tree was carried out, and the physicians in charge of the case were quite satisfied that they had done an adequate repair.

The following morning, on Jan. 10, his condition was quite satisfactory, considering the magnitude of the injury. X-ray showed the left lung to be completely reexpanded; there was no egress of air through the underwater seal trap, and the patient had regained consciousness. His respirations were quite noisy, and bronchoscopic aspiration was again carried out, with a vast improvement in his breathing. All that day he maintained a normal blood pressure in the neighborhood of 120 to 130 systolic over 60 to 80 diastolic. By evening, however, he became somewhat disoriented and there was a gradual rise in his blood pressure.

By the following morning, Jan. 11, which was two days after his accident, he was quite comatose and carried a blood pressure of 200 systolic. The physicians in charge of the case wondered about cerebral damage incident to his accident, what with the increasing unconsciousness and the rise in

Fig. 2.—Trachea and main bronchi. Pointer indicates region of repair. Specimen opened posteriorly.

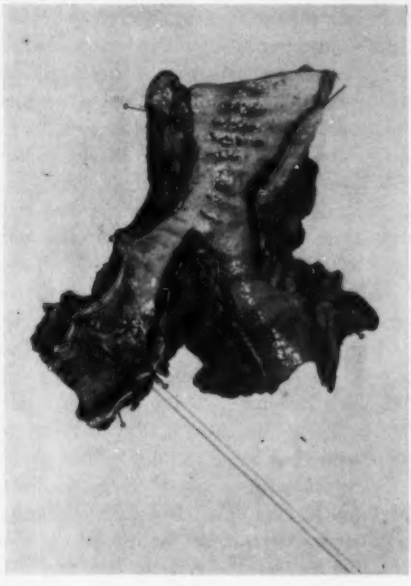

Fig. 3.—Aorta with probe through point of rupture. Note damage completely encircling this structure. Interior view.

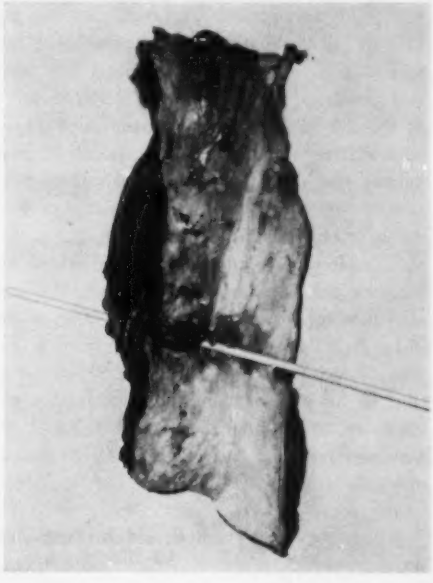

Fig. 4.—Aorta with probe in point of rupture of aneurysm. Exterior view.

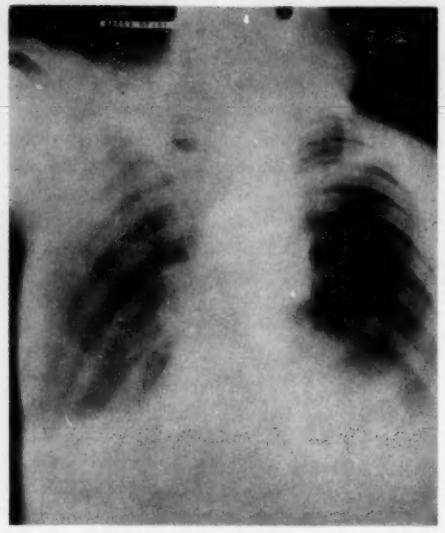

Fig. 5.—Preoperative x-ray.

blood pressure. However, a neurosurgical consultation pointed out that the patient also had an extremely rapid pulse, which would not go with an intracranial lesion of expanding nature. A spinal tap showed grossly bloody spinal fluid under no pressure. It was the neurosurgeon's opinion that the log falling on the patient's back damaged the neural arch and had traumatized the cord and produced the blood. He felt that some of this blood was going cephalad and might be irritating the cortex to the point of producing the unconsciousness. Another possibility suggested by the neurosurgeon was some embolic phenomenon to the brain.

The blood pressure remained elevated all that second day and on into the night and well into the following early morning, when it began to drop gradually. By 6:30 a. m. just two and three-fourths days since the time of his accident, the systolic blood pressure fell to 90 and before blood could be administered and other resuscitative measures instituted, the patient quite suddenly died.

An autopsy was granted, and the examiner was prepared to demonstrate some intracranial lesion. However, his curiosity was satisfied after he had examined the chest and abdominal cavities. For in both pleural cavities there was an excess of 500 cc. of fresh blood, and there was evidence of ecchymosis and dissecting hemorrhage all up and down the mediastinal and retroperitoneal tissues postoperatively. Dissection revealed that the anastomosis of the bronchus had held well, though the man had a purulent tracheobronchitis as would be expected. The cause of death was found to be the rupture of a false aneurysm of the aorta. The cause of the false aneurysm was an almost complete transection of the aorta at the same level at which the bronchus was injured. After all the viscera had been removed from the body, the cause of both injuries was evident, for there had been a complete fracture of the thoracic spine between the fifth and sixth vertebrae. Prior to death a portable x-ray of the chest had been taken for bone detail, but being technically not perfect failed to reveal any injury to the bodies of the thoracic vertebrae. It did show damage to the neural arch in the vicinity of the blow from the log, but the vertebral column itself

seemed intact. However, at autopsy, one could easily move the two segments of the thoracic spine, one upon the other, and undoubtedly the blow from the log had caused a full rupture of the annulus and disc between the fifth and sixth thoracic vertebrae, causing the one segment to move forward and shear both the bronchus and the aorta. Undoubtedly, the shearing of the aorta was not complete, and there must have been a few fibers remaining which obscured the aortic injury at the time of thoracic surgery. However, the injury was of sufficient magnitude that the force of the pulsating blood dissected through the damaged portion of the aorta and formed a false aneurysmal sac. Undoubtedly for a time this compressed the aorta, causing the elevated blood pressure. Then finally the sac of the false aneurysm ruptured, with immediate fatality.

The physicians in charge of this case were no little chagrined at the autopsy finding for, certainly, had this injury been suspected at the time of surgery, attempt should have been made to repair the injured aorta, and this would have been possible, had the injury been recognized.

It should also be mentioned that this case was interesting from an entirely unrelated point of view. Some eight years ago, this man had had an almost complete gastric resection with the excision of a portion of the left lobe of the liver for a lymphosarcoma of his stomach, from which he completely recovered, and at the time of autopsy absolutely no vestige of his original disease was evident.

Green Clinic, 709 Vienna St.

Fatal Rupture of an Ivalon (Polyvinyl Formalinized) Sponge Aortic Graft into Duodenum

A Case Report

LOWELL BROWN, M.D., and HARRY ESSIG, M.D., Freeport, L. I., N. Y.

One of the major problems in vascular surgery is the need for satisfactory blood vessel substitutes. Because of the difficulties in procurement of arterial homografts as well as certain defects in their behavior, and because of the limited usefulness of autografts, search continues for a satisfactory synthetic arterial replacement.

This case is being reported because it illustrates dangerous deficiencies in a type of arterial replacement which has seen fairly wide use and which in fact is still occasionally being commented on favorably in the literature.[1]

Report of Case

A 74-year-old white man was admitted to Meadowbrook Hospital on June 13, 1956, with the chief complaint of vague right-sided abdominal pain of 24 hours' duration, becoming increasingly severe and associated with several episodes of emesis. He had been in good health until the present episode. Shortly prior to admission he had taken an enema and collapsed.

Examination revealed an alert, pale, white man with a blood pressure of 80/50 and a pulse of 80. Pertinent findings consisted of right-sided abdominal tenderness extending into the flank. There was also right costovertebral angle tenderness. A 12 cm. mass was felt in the lower midabdomen, which showed an expansile pulsation. The extremities were warm, and all pulses were palpable bilaterally. Laboratory examination revealed a hemoglobin of 10.0 gm. and a hematocrit reading of 32. Urinalysis was not remarkable. X-ray of the chest was within normal limits. An abdominal film showed calcification of the aorta and obliteration of the right psoas shadow.

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From the Vascular Service, Meadowbrook Hospital, Hempstead, N. Y.

A diagnosis of ruptured abdominal aortic aneurysm was made. Transfusions were started, and a catheter was placed in the bladder. Papaverine, ½ grains (32.5 mg.), was given intramuscularly. The blood pressure shortly rose to 120/80, and he began to secrete urine.

Operation was now commenced under fractional spinal anesthesia. An aneurysm of the abdominal aorta was found measuring approximately 12 cm. in diameter, with extensive retroperitoneal hemorrhage. The superior limit of the aneurysm was found to be 3 cm. below the renal arteries, while the lower limit was just above the bifurcation.

The aorta was cross clamped below the renal arteries, and both common iliac arteries were clamped. The aneurysm was then excised. Dilute heparin was instilled proximally in the aorta and distally into both iliac arteries. A sleeve graft of Ivalon (polyvinyl formalinized) sponge was used to bridge the defect. The graft was constructed by wrapping 3 mm. strips of sliced sponge over a tubular aluminum stent. The Ivalon was then compressed by tight gauze wrapping and autoclaved for 20 minutes.[2] The finished graft measured about 3 mm. in thickness. Running over-and-over 0000 arterial nonabsorbable (silk) surgical sutures were used for the anastomoses. Good pulsations in both iliac arteries were noted on removing the clamps. A moderate amount of bleeding occurred through the interstices of the graft, but this subsided rapidly with gentle pressure.

Immediately postoperatively, good pulses were felt throughout the right leg. On the left, however, the femoral, popliteal and dorsalis pedis pulses were not palpable. Despite this, the leg was warm and of good color. Approximately two hours later all pulses were felt bilaterally.

The aorta had been clamped for 2 hours 37 minutes and the total operating time was 4 hours 15 minutes. A total of 5,000 cc. of blood, 1,000 cc. of plasma, and 1,200 cc. of dextrose and water were given.

The postoperative course was uneventful and he was discharged on the 15th postoperative day to the outpatient clinic. At follow-up he continued to have bilateral dorsalis pedis pulses, and it was

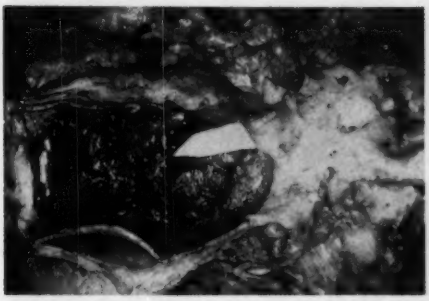

Fig. 1.—The opened Ivalon prosthesis is shown with its two intact aortic anastomoses. The surrounding envelope of fibrous tissue can be seen as it peels easily from the Ivalon. An arrow illustrates the site of rupture.

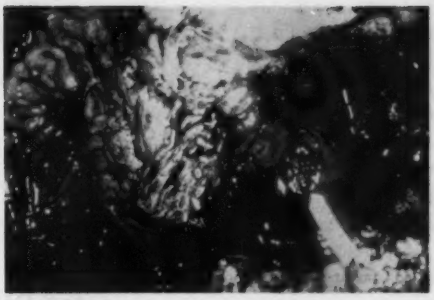

Fig. 2.—The opened duodenum is shown, with an arrow indicating the site of rupture. A massive thrombus lies adjacent in the duodenum.

noted that he could walk up to one mile without claudication and that his feet, which had been cold for several years, were now warm.

On Aug. 15, 1957, at 2 a. m., 14 months after operation the patient's wife was awakened by his labored breathing. He was unresponsive and shortly vomited a large quantity of bright red blood. He was taken to Huntington Hospital by ambulance and died en route.

A postmortem examination was performed by Dr. Cares with the following findings: The stomach, duodenum, and small intestines contained large amounts of fresh and clotted blood. There was no blood in the peritoneal cavity or in the retroperitoneal spaces. The Ivalon sleeve graft (Fig. 1) was surrounded by an envelope of fibrous tissue and was not grossly "invaded" by this tissue. Located on the right anterior aspect of the upper third of the prosthesis was a defect communicating with the third portion of the duodenum. There was a 3 cm. mass pouting for 1 cm. above the level of the duodenal mucosa, in the center of which was a 1 cm. crater-like opening (Fig. 2). A probe demonstrated the communication between the lumen of the prosthesis and the duodenum. A 4 cm. area of thrombus was present on the prosthetic side of the communication, and a 3 cm. thrombus lay free in the duodenum just distal to the site of rupture. Further examination of the prothesis revealed its walls to measure quite uniformly 3 mm. in thickness. In appearance it was light pink and grossly resembled such grafts seen at autopsy a week or two after insertion. The lumenal aspect was smooth and glistening. At the site of rupture there was an absence of prosthesis for area of 3 to 4 cm. In diameter and at the margins of this defect the prosthesis was attenuated from its 3 mm. diameter to mere shreds. This attenuated margin formed a rim of 1 cm. radius.

The aorta above showed moderately advanced atherosclerotic change. The proximal suture line was 13 mm. from the right renal orifice. Both renal vessels were patent. Distally there was an aortic cuff measuring 1 cm. Both common iliac vessels were widely patent, although again they showed moderately advanced atherosclerotic change. The cause of death was rupture of the Ivalon sleeve graft into the third portion of the duodenum.

Comment

Rupture of aneurysms into the duodenum have been reported on a number of occasions. Skromak and associates [3] report two such instances together with a review of the literature. Voyles and Moretz [4] report resection of an aneurysm following perforation into the duodenum with immediate success but with death on the 39th postoperative day from complications following delayed hemorrhage from an iliac anastomosis.

We have had three such experiences with rupture of aneurysms into the duodenum at our hospital. Certainly with massive gastrointestinal bleeding we should consider aneurysm in the differential diagnosis.

Ohara and Nakano [5] have recently reported the rupture of a plastic aortic graft of Amylan (nylon 6) fused with polyethylene into the duodenum, resulting in fatal hemorrhage one year after implantation following resection of abdominal aortic aneurysm.

Clayton and associates [6] have reported a most unusual instance of massive hemorrhage into the small intestine caused when

the iliac anastomosis of a bifurcation graft of nylon eroded the bowel. The graft had been inserted 17 months before, after resection of a huge abdominal aortic aneurysm. This patient, unlike ours, had several such episodes over a period of four weeks prior to the final terminal episode and might therefore very possibly have been saved by an early operation.

Shumacher[7] has reported a most significant observation on the importance of maintenance of tensile strength by an aortic replacement graft. He had utilized a fused Dacron-polyethelene bifurcation graft to replace an abdominal aortic aneurysm. Subsequently thrombosis of one iliac artery occurred. At operation the thrombus was removed. Also in the course of the procedure the entire bifurcation graft was removed through the iliac arterotomy, leaving only the surrounding fibrous envelope. All pulses were restored. The patient was then well until two years later, when he had to be reoperated on for recurrence of the aneurysmal dilatation of the abdominal aorta. Our experience would seem to lend further emphasis to the thesis that the strength of a vascular replacement is dependent on the maintenance of the tensile strength in vivo of the graft material.

Summary

A case of ruptured abdominal aortic aneurysm was treated with initial success by a graft of Ivalon (polyvinyl formalinized) sponge. Fourteen months later fatal rupture of this graft occurred into the third portion of the duodenum. The rupture occurred well away from the suture line. There was diffuse thinning of the graft about the area of rupture. A fibrous envelope was present surrounding the graft, but there was no significant gross invasion of the interstices of the graft.

It is felt that this case lends support to the concept that the strength of a vascular replacement is dependent on the maintenance of the tensile strength in vivo of the graft material. The use of Ivalon as a vascular graft with present techniques is open to grave question.

Medical Arts Building, 155 W. Merrick Rd.

REFERENCES

1. Fitch, E. A., and Denman, F. R.: An Adjustable Aortic Graft, A. M. A. Arch. Surg. 75: 1027, 1957.

2. Ayers, W. B.; Cincotti, J. J., and Gliedman, M. L.: Experiences with Ivalon (Polyvinyl Formalized) Sponge Prostheses for Vascular Replacements, A. M. A. Arch. Surg. 74:173, 1957.

3. Skromak, S. J.; O'Neill, J. F.; Ciccone, E. F., and Snyder, R. J.: Aneurysm of the Abdominal Aorta: Two Cases with Rupture into the Gastrointestinal Tract, Gastroenterology 33:575, 1957.

4. Voyles, W. R., and Moretz, W. H.: Rupture of Aortic Aneurysms into Gastrointestinal Tract, Surgery 43:666, 1958.

5. Ohara, I., and Nakano, S.: Rupture of Arterial Plastic Prosthesis (Amylan-Polyethylene Tube): Report of a Case and Experimental Studies, A. M. A. Arch. Surg. 77:55, 1958.

6. Clayton, H.; Birch, L.; Cardwell, E. S., and Zimmerman, S. L.: Suture-Line Rupture of a Nylon Aortic Bifurcation Graft into the Small Bowel, A. M. A. Arch. Surg. 73:947, 1956.

7. Shumacher, H. B.: Plastic Grafts for Aortic Substitution in Man, A. M. A. Arch. Surg. 75: 413, 1957.

Intranasal Encephalomeningoceles Associated with Cranium Bifidum

HENRY W. DODGE Jr., M.D.; J. GRAFTON LOVE, M.D., and JAMES W. KERNOHAN, M.D., Rochester, Minn.

The recommendation of innumerable surgical procedures for a given condition is prima facie evidence that ultimate cure is difficult to attain. This has been especially true in the development of surgical techniques for the repair of anteriorly located encephalomeningoceles. It is our purpose to present an outline of the already well-known basic facts about these intriguing and difficult surgical lesions; to consider briefly a number of types of surgical repair previously reported, and then to describe our method of operation.

Extracranial prolongation of brain tissue with its non-nervous coverings, the encephalomeningocele, occurs usually in the midline of the cranial vault at the frontal or occipital poles. The great majority of encephalomeningoceles are situated in the occipital region, while only about 15% to 20% are nasofrontal or anterior in location. Lateral herniation apparently is extremely rare and is said by some not to have been observed.

The incidence of encephalomeningoceles has been estimated at about one in every 3,500 to 4,000 births, although presumably medical consultation is not required in a certain number of instances. Obviously in order to have cerebral tissue present outside the skull, a defect must exist in the medial bony plate, and this defect is commonly designated "cranium bifidum" (Fig. 1).

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Section of Neurologic Surgery (Drs. Dodge and Love); Section of Pathologic Anatomy (Dr. Kernohan), Mayo Clinic and Mayo Foundation. The Mayo Foundation, Rochester, Minn., is a part of the Graduate School of the University of Minnesota.

Fig. 1.—Child suffering from a basal encephalocele passing down through a cleft palate and issuing from the mouth. (Modified from Virchow, R.: "Die krankhaften Geshwulste," Berlin, 1863, Vol. 1, p. 188.)

Over the years this relatively small number of cases has received continued attention from surgeons with sporadic flurries of intense interest, due in the main to the spectacular and lethal consequences of lack of recognition of the nature of the lesion and of inadequate surgery. The literature is replete with warnings about innocent-appearing polyps lying high in the nose, or small soft tumors about the root of the nose and inner canthus of the eye (Fig. 2).

Excellent comprehensive reviews of the subject of congenital anterior herniation of the brain have been published by Anderson,[1] Mood,[2] Gisselsson,[3] Moore,[4] McGillicuddy,[5]

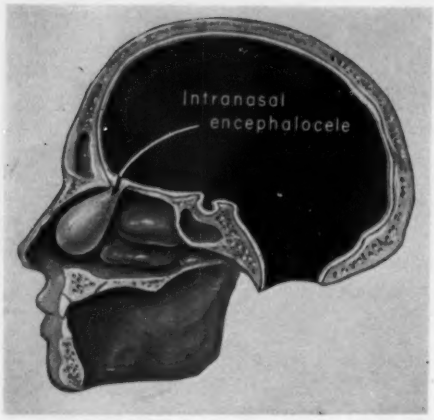


Fig. 2.—Sagittal view of basal type of encephalocele passing from cranial cavity into the nose.

and others, while interesting statistical analyses of the subject have been presented in articles by Schwidde,[6] Ingraham and Swan,[7] and Murphy.[8]

Classification of Encephalomeningoceles

For practical purposes in the past, anterior herniations of the brain have been divided into sincipital herniations, or those presenting visible evidence of their existence externally, and basal herniations, or those not presenting externally but passing into the nasopharynx, orbit behind the globe, and the mouth (Table). The classification in the Table is probably the one most widely accepted over the past years.

In employing the word "hernia" to describe the existence of brain tissue outside of the cranial cavity, we recognize immediately that there is legitimate argument against the term, since the mass encountered extracranially may not be an actual herniation of the brain, but may have remained outside while the skull proper was being formed. Although there are objections, the expression "anterior cerebral hernias" for the encephalomeningoceles located nasofrontally has gained validity through long usage in the literature (Fig. 3).

Etiologic and Pathologic Aspects of Anterior Encephalomeningoceles

Many authors have speculated on the etiologic aspects of anterior encephalomeningoceles. Most agree that they are developmental, nonhereditary, nonfamilial, and not necessarily associated with other congenital defects. In general, the theories fall into two main groups: those postulating

Anterior Cerebral Hernias

Basal type (Heineke)
In the basal type of anterior cerebral hernia there is no externally visible tumor.
- Encephalocele sphenopharyngea
  The pedicle of the hernia passes through an opening between the sphenoid and ethmoid bones or through one of these bones into the nasopharynx.
- Encephalocele spheno-orbitalis
  This type of hernia exists through the superior orbital fissure to enter the orbit behind the ocular globe.
- Encephalocele sphenomaxillaris
  This type of hernia passes through the superior orbital fissure, but instead of remaining in the orbit descends through the inferior orbital fissure into the sphenomaxillary fossa. The tumor may be palpated in the patient's mouth.

Sincipital type (Von Meyer)
The sincipital type of anterior cerebral hernia forms externally visible tumors in the nasofrontal regions of the head.
- Encephalocele nasofrontalis
  The pedicle of the hernia passes between the frontal and nasal bone to form a swelling in the midline in the region of the glabella.
- Encephalocele nasoethmoidalis
  This type of hernia passes between the frontal and nasal bones on one side and the lateral mass or labyrinth on the other. The mass appears externally in the region of the border between the osseous and cartilaginous portions of the nose, hanging down toward the tip or wing of the nose.
- Encephalocele naso orbitalis
  The pedicle of this hernia passes between the frontal, ethmoid and lacrimal bones, enters the orbit, and presents at or near the inner canthus of the eye.

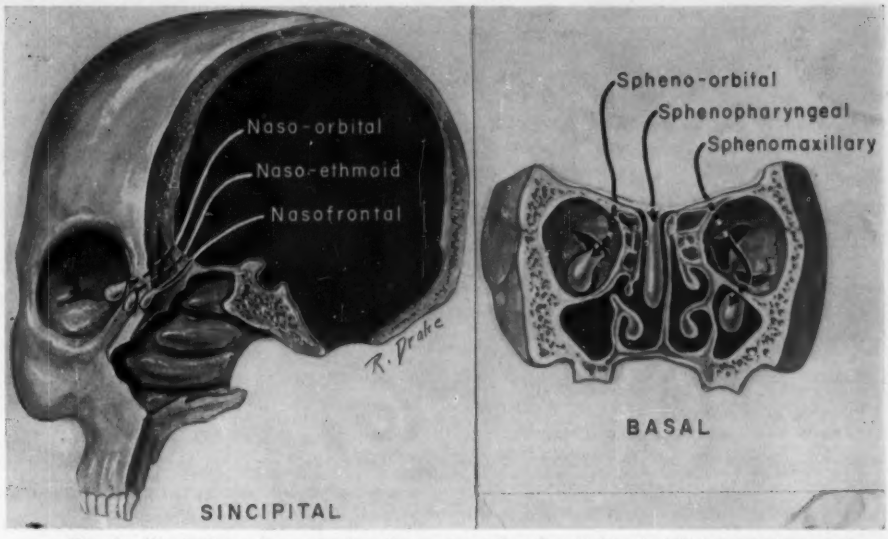


Fig. 3.—Illustrations demonstrating the routes taken by (left) sincipital herniations and (right) basal herniations of the brain. (See Table for detailed classification of Von Meyer and Heineke.)

a failure of the ethmoid plate to form about a bundle of olfactory nerves, therefore leaving a defect through which herniation of the brain subsequently takes place, and those postulating a preexisting encephalocele due to failure of the anterior neuropore to close and separate from the skin which prevents the ingrowth of mesoderm, a precursor of the ethmoid bone. The latter theory, which is known as "St. Hillare's adhesive theory," is favored by the observation that in the occasional case bone may pinch off the stalk of the encephalocele and leave an isolated cyst within the nasal cavity. This theory received further strengthening from Sternberg's [9] observations in 1929.

Concerning congenital anterior encephaloceles, the question has frequently arisen and much controversy has occurred as to the coexistence of the so-called nasal glioma. Best evidence suggests that tumors of true neurogenic origin may be found on the bridge of the nose, at the base of the nose between the eyes, or within the nasal cavity itself. Some of these tumors are not neoplasms but are heterotopic masses, while others are neoplasms with power to spread, destroy bone tissue, and metastasize. These neurogenic masses may be divided into two groups: (1) those on the bridge or at the base of the nose or over the glabella, and (2) those within the nasal cavity, conforming to the classification of the anterior cerebral herniations themselves (Table).

As Davis [10] has pointed out, the first group are usually heterotopic glial masses made up of normal astrocytes within a mesh of connective tissue. These usually are not neoplasms, although recently one of us had the opportunity of observing one that had recurred on two occasions and had extended into the upper eyelid. Some of these heterotopic nodules are enclosed in meninges and, according to Schmidt, probably represent an encephalocele, although Süssenguth [11] thought that they were of olfactory origin. In this same region neoplasms of meningeal origin have been found, and they are usually meningiomas of a low degree of malignancy. These tumors probably arise from arachnoid cell clusters in meningoceles, which form while the bones of the skull in this region are being closed. Sometimes these meningoceles maintain a

free communication with the subarachnoid space; more often there is no communication, and occasionally only a cord of obliterated meninges remains. Within the nasal cavity neurogenic tumors are of a different type. Berger and Coutard [12] first described these tumors as "esthesioneuroepitheliomas," and two years later they described another type of variant which they called "esthesioneurocytoma." Recently Mendeloff [13] described 6 more cases and found 14 others in the literature.

The first of these two types, the esthesioneuroepithelioma, has "round and oval spaces lined by tall columnar cells forming true rosettes," whereas the esthesioneurocytoma has "rests of undifferentiated tumor cells without architectural arrangement." Both types of tumor tend to invade the surrounding tissue and destroy the bone. Only the esthesioneurocytoma has been found to metastasize to regional lymph nodes.

Recently one of us (J. W. K.) had the opportunity of examining a section of a polypoid tumor of the nose submitted by Dr. D. A. de Santo of San Diego, Calif. This tumor had been located high in the posterior part of the nasal cavity and consisted of islands of adult type of astrocytes surrounded by dense connective tissue. There was no evidence of cellular activity, and it was considered to be a mass of heterotopic glial tissue made up exclusively of astrocytes. Thus, it is not remarkable that the nature of cellular masses found in association with these anterior cerebral herniations has been the subject of controversy.

Diagnosis

A congenital tumor in the region of the base of the nose, if it is small, may suggest the presence of one of the lesions just described, or of a dermoid cyst, a mucocele, or an encephalomeningocele, and therefore surgical intervention should be undertaken with caution. Basal forms of cerebral hernia, that is, the "hidden" types, may be easily mistaken for benign nasal polyps, or when seen in the epipharynx may be confused with other intrinsic tumors.

The problem of diagnosing an encephalocele of the sincipital variety, which is externally visible, is relatively simple (Fig. 3, left), once one thinks of the possibility. However, the problem of arriving at a diagnosis for the basal type (Fig. 3, right), that is, the encephalomeningocele which passes through into the orbit behind the globe, into the nose, the nasopharynx, or mouth may be difficult. Most authors have emphasized certain points concerning the intranasal forms which seem worthy of repeating.[3]

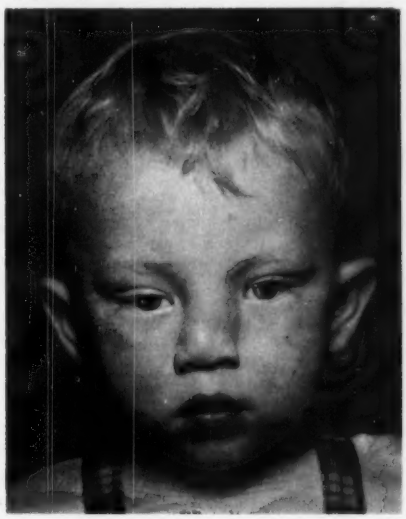
Usually the mass in the nasopharynx or nose (Fig. 4, *a* to *e*) is encountered at birth, or symptoms arising from the mass are noticeable in early childhood. In a number of cases, respiratory obstruction is troublesome relatively early in the child's development. If the nasal tumor is an encephalocele, close examination will reveal the mass to issue from the superior part of the nasal septum, and the nasal mucous membrane appears to be continuous over the entire tumor. The finding of a nasal polyp in an infant or young child should be given more than cursory attention, since nasal polyps are uncommon in young children. Another interesting point, frequently mentioned in the examination of patients harboring these lesions, is widening of the nasal root, or an unusually large distance between the patient's eyes.[3] This should not be confused, however, with hypertelorism, a separate skull condition (Fig. 5).

In the investigation of an intranasal polyp certain signs should suggest that this tumor may be in communication with the intracranial portion of the nervous system. If the "polyp" is an encephalomeningocele, it may be seen to fluctuate synchronously with the patient's pulse or respiration; or if the patient is caused to strain or the examiner manually obstructs the jugular veins in the neck, the tumor may swell or even leak cerebrospinal fluid. However, if the "polyp"

Fig. 4.—Examples of more pronounced types of sincipital encephalomeningoceles. (*a* to *d*) Nasal herniations. (*e*) Orbital herniation. (*a*, *b*, and *c*, Mayo Clinic cases. *d*, Reprinted from Pinto, F.; Pinto, A., and Moscoso, A.: Meningoenfaloceles nasais, *Rev. brasil. cir.* 33:84-87 [Jan.] 1957, and from "Year Book of Neurology, Psychiatry and Neurosurgery" [1957-1958 series] Chicago, The Year Book Publishers, p. 442. *e*, Reprinted from Rand, C. W.: "The Neurosurgical Patient," Springfield, Ill., Charles C Thomas, Publisher, 1944, courtesy of publisher.)

under suspicion has become fibrosed or the pedicle is partially obliterated, these characteristics may be absent. Many have had an unpleasant experience with the small encephalocele situated in the nose that did not respond to these diagnostic measures, and yet, on transnasal removal, cerebrospinal fluid leaked copiously from the amputated stalk. On the other hand, in the presence of ordinary nasal polyps there may be an associated rhinorrhea which can be disconcerting.

Fig. 5.—Young patient showing the widened nasal root, increased interpupillary distance, and nasal bulge often described as significant clues to correct diagnosis. The partial aniridia in this case was coincidental.



If the nasal discharge consists of cerebrospinal fluid, laboratory examination is diagnostic. Commonly such fluid is colorless, limpid, and slightly viscous, and has a specific gravity between 1.004 and 1.008. Traces of protein and small quantities of inorganic salt and dextrose may be found, and the cell count may vary from 5 to 10 cells per cubic millimeter. However, in actual practice, it is frequently sufficient to determine the sugar content alone. The normal amount of sugar in the cerebrospinal fluid varies between 45 and 80 mg. per 100 cc.

Should one still be in doubt about the nature of the discharging nasal fluid, a small quantity of indigo carmine, 1 cc., may be introduced into the spinal subarachnoid spaces, and if the nasal discharge is cerebrospinal fluid, it will be seen to have attained a bluish-green color when tested on a white background.

One confusing aspect, however, has been added by Ichelberger and Linsey, and later Lillie,[14] who reported elevated sugar and chloride content in fluid aspirated from true primary intranasal cysts. It was their impression that this fluid could be differentiated, however, from cerebrospinal fluid by properties of the fluid other than the sugar and chloride content.

Occasionally roentgenographic examination of the patient's head may give valuable diagnostic aid by demonstrating a defect in the anterior basal part of the skull. In our experience the most satisfactory roentgen views to demonstrate such defects are the following: routine roentgenograms of the skull (views obtained in the posteroanterior, Caldwell, anteroposterior, Towne, and stereolateral positions), stereoscopic views of the base of the skull (submentovertical); tomograms obtained in posteroanterior, basal, or lateral positions, stereoscopic views obtained in the Water position, and occasionally projections obtained in the Rhese position for the optic canals.

The complexity of diagnosis is increased when one is confronted with an intranasal tumor high in the nose which does not respond to pressure on the jugular veins or various other tests to increase intracranial pressure, which does not fluctuate synchronously with the pulse and respiration, and which does not leak spinal fluid. In some cases this may be a rather fibrous remnant of an encephalomeningocele, and undoubtedly in many cases this type of lesion has been removed through the nose with impunity. However, it may be a small encephalomeningocele communicating freely with the subarachnoid spaces, and resection through the nose may lead to profuse cerebrospinal-fluid rhinorrhea and the threat of fulminating meningitis. For this reason it seems reasonable to suggest that the care of these patients be the responsibility of a team of surgeons. This will be alluded to later, and the occasional diagnostic dilemma offered by this small, high-lying lesion brings up the quickly to be condemned idea of aspiration or excision as a means of diagnosis.

Various Methods of Surgical Treatment

Apparently Richter,[15] in 1813, was the first author to report a clinical case of nasofrontal encephalomeningocele. This was encountered in a man, 60 years of age, who subsequently died from infection. The first successful operation was said to have been performed in Fenger's [16] case in 1895, although a Russian author stated in 1952 that the operation had been performed 70 years before in Russia.

There seems to be no dispute among surgeons about the difficult and potentially lethal nature of the nasal encephalocele. The high postoperative death rate has been chiefly the result of ascending infection resulting from the cerebrospinal fluid fistula at the site of inadequate dural repair.

In the intervening years many surgical procedures have been evolved for the cure of anterior encephalomeningoceles, but they are all basically accomplished by one of three routes, or a combination thereof: (1) external, via the root of the nose; (2) transnasal, and (3) intracranial. Regardless of the route employed, however, the chief problem has been, and still remains today, the closure of the dural defect following amputation of the encephalocele. This seems to be an extremely important point, since many of the operative procedures designed in the past have seemingly placed more emphasis on the repair of the bifid skull than on the dural defect.[17]

Obviously when the pedicle or stalk of the encephalocele is narrow and the skull opening is small, once satisfactory ligation of the pedicle has been achieved, there would appear to be little necessity for plastic repair of the skull. However, with larger encephaloceles and correspondingly greater skull openings, increasing attention must be given to the defect in bone.

Whenever amputation of the larger encephalomeningoceles has precluded primary dural closure, substitute techniques have

been quickly devised. One of the simplest methods described is the releasing counter incision in the dura of the bifrontal cerebral convexity, preserving the arachnoid and allowing the subfrontal dura to be more easily approximated. Sliding dural autografts, fascia lata, and, more recently, preserved human dura have been utilized for water-tight repair.

In some cases, human muscle has been included in the suture line of the repair, or an iodoform gauze wick has been placed in the subfrontal dural defect to stimulate the growth of scar tissue. Some have found animal peritoneum (Luken's membrane) helpful as a neodura.

The advent of the newer synthetic plastic materials into surgery gave rise to the use of polyethylene film and polyvinyl sponge for the replacement of missing segments of dura. That no one of the previously mentioned substances has been entirely adequate for the purpose designed is attested to by the number of different materials advocated for the achievement of a water-tight dural repair. Actually many surgeons have used a combination rather than depend on any one material or method alone.

Consideration of the treatment of the bifid skull, once good dural repair or tight stalk ligation has been carried out, in many cases seems only of secondary importance. However, the larger dehiscences in the skull have provided the inspiration for much of the surgical ingenuity reported in the literature. Earlier articles advised bone grafts to the basofrontal skull defect. Some of these grafts were free grafts taken from skull, tibia, ilium, and ribs; others were muscle periosteal bone grafts swung as a flap from the skull of the same patient. These were often traumatic and deforming, and many patients died as a result. Lately, the possibility of using preserved bone from a bone bank has become a reality. Grafts have also been devised from strips of periosteum alone, and from preserved cadaver cartilage,[18] while certain of the smaller basofrontal defects have been plugged with Horsley's wax, absorbable gelatin sponge (Gelfoam), or oxidized cellulose (Oxycel). Figi described the interesting use of a skin graft over a plastic stent after ligation and repair of the stalk.

The use of tantalum screen, and Vitallium tantalum, or stainless steel plates has been helpful in a number of patients in whom the larger defects in the skull predisposed to further prolapse of brain tissue. Of the plastics which can be molded at the operating table, methyl methacrylate in mixed monomer and polymer forms has been used successfully. Premolded plates and bolts of Plexiglas or polyethylene also have found favor. Plexiglas is the subject of a number of more recent Russian articles.

Reviewing the various methods for the cure of anterior cerebral hernias led us to conclude that three cardinal points are worthy of reemphasis. These are (1) accurate preoperative diagnosis of the type of lesion and its extent; (2) adequate water-tight repair of the affected dura, and (3) if required, simultaneous cranioplasty.

To suggest that the transcranial repair of encephaloceles by the neurosurgeon is the only answer to these difficult cases would be erroneous. Many experienced general, nasal, and plastic surgeons are familiar with this area of the skull and brain and have achieved brilliant results transnasally or by the external route through the face. Past experience has suggested, however, that despite the approach used, the initial surgical effort should be directed toward ligation of the stalk of the encephalocele and isolation of the subarachnoid pathways of the nervous system by water-tight dural closure to prevent infection. Secondary attention should be given, only then, to the skull defect and the extracerebral mass. The use of prophylactic antibiotic precautions seems a must.

Our Method.—In our practice the intracranial method has offered the following advantages: (1) concealed intrahairline scalp incision; (2) excellent visualization of the subfrontal lesion; (3) adequate room

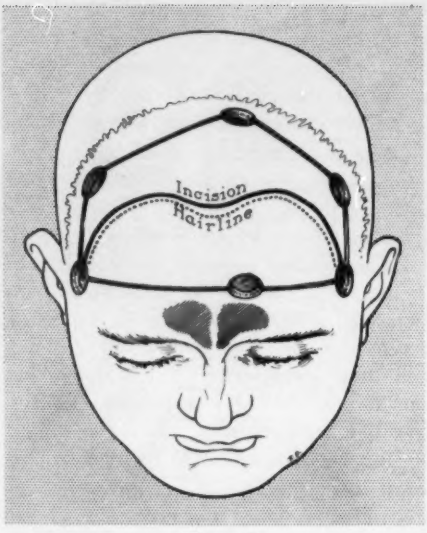


Figure 6

Fig. 6.—Curving intrahairline coronal scalp incision and bifrontal craniotomy frequently employed for transection and repair of anteriorly located encephaloceles.

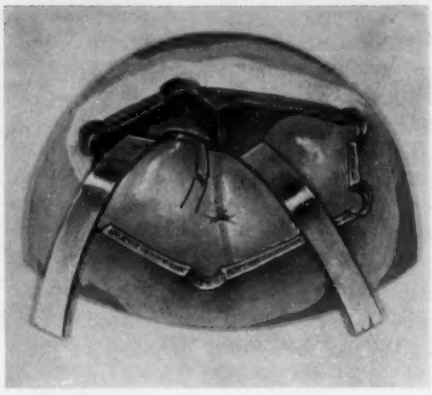


Figure 7

Fig. 7.—Surgeon's view of basal type encephalomeningocele utilizing bifrontal exposure. The frontal lobes are retracted with the aid of removal of small amounts of cerebrospinal fluid by means of an indwelling lumbar spinal catheter. Note purse string ligature around the stalk of the encephalocele.

for careful dural repair, and (4) opportunity to perform simultaneous cranioplasty in a relatively uncontaminated field (Fig. 6).

After adequate preparation of the patient with the antibiotics, usually a unilateral frontal or a bifrontal craniotomy is employed to approach the pedicle of the encephalomeningocele from the cranial side. Visualization of the cribriform area can be aided by the removal of a small amount of cerebrospinal fluid through an indwelling lumbar spinal catheter. This allows for easier retraction of the frontal lobes. To be emphasized for patients with cerebrospinal-fluid rhinorrhea is the necessity for an intratracheal airway with a cuff or some protection against aspiration of fluid while under anesthesia. For children with a high degree of upper respiratory obstruction, preliminary tracheotomy has been recommended, with sound evidence to substantiate its efficacy.

Once the skull defect has been identified, and the base or stalk of the encephalocele isolated, simple ligation and section may be adequate. If the base of the encephalocele is large, the resulting dural defect may require careful dural repair. Should primary suture of the dura be impossible, we have most recently favored the use of preserved human dura to achieve the necessary watertight closure (Fig. 7).

For smaller defects in the skull no further repair seems necessary after the dural closure has been achieved. However, if the corresponding defect in the skull is large, stainless steel, tantalum, or acrylic have seemed to us most satisfactory for the basal cranioplasty. This has been true both from the standpoint of ease of handling and lack of trauma to the frontal lobes in performing the cranioplasty. Equally as important, these materials have proved to be kindly accepted by the patients' tissues, causing no apparent foreign-body reaction.

Quite obviously the transcranial repair of an anterior encephalocele does not allow for the treatment of the nasal tumor. Certainly any attempt to deliver the nasal portion of the encephalocele through the cranial exposure should be strongly decried.

Many of the smaller encephaloceles in the nose or nasopharynx will atrophy after

intracranial repair, and ultimately slough out, requiring no particular attention. For the more massive types, or if considerable nasal or facial deformity exists, secondary external operation by a nasal or plastic surgeon may be necessary. Once water-tight intracranial repair of the stalk of the encephalocele has been achieved, the time and method of treating the extracerebral mass or deformity must be dictated by the special circumstances in the individual case.

Before operation is advised for massive cerebral herniations in newborn infants with communication to the ventricular system and wide cranial dehiscences, the probability of achieving ultimately a relatively normal person should be carefully considered.

Comment.—Our experience indicates that in the proper surgical treatment for the potentially lethal nasofrontal encephalocele the primary attack should be directed at amputation of the base or stalk intracranially with careful water-tight repair of the dura by one means or another. The bifid skull, unless the dehiscence is large, is of only secondary importance, while the treatment of the nasal mass, unless a high degree of respiratory obstruction exists, is of relatively minor import.

If facial deformity or a large nasal or nasopharyngeal tumor is present, or should some special problem such as a cleft palate intervene, we believe that another member of the team (the nasal or maxillofacial surgeon) should recommend and carry out the definitive measures at an appropriate time. Seemingly in cases so managed and with the help of antibiotic therapy, the once alarming morbidity and mortality associated with these anterior encephaloceles should be virtually abolished. In addition, present-day surgeons should anticipate for the patient a pleasing cosmetic result (Fig. 8).

Summary

Anterior nasofrontal encephalomeningoceles may be basal (hidden) or sincipital (externally visible). They are developmental, nonhereditary, nonfamilial, and not necessarily associated with other congenital defects; they occur approximately once in every 3,500 to 4,000 births and are associated with a bifid skull. Encephaloceles of the nose are commonly mistaken for polyps. Although nasal polyps are infre-

Fig. 8.—Two patients after transcranial resection and repair of nasal encephalomeningoceles, (*a*) 1 year and (*b*) and (*c*) 12 years after operation. Note lack of surgical scar due to hairline incisions.

quent in children, anterior cerebral herniations are most likely to be encountered in the younger age groups.

The externally visible encephaloceles vary from large deforming masses to small soft swellings near the bridge of the nose. Basal types of hernias may be silent, result in cerebrospinal-fluid rhinorrhea, or because of size and location cause respiratory obstruction.

In the absence of obvious deformity of the face certain signs, such as a broadened nasal root and widened intrapupillary distance, have been reported often in patients harboring these lesions. Because of the communication of the stalk with the central nervous system, surgical removal in the past has been attended by a high morbidity and mortality from infection. Roentgenograms of the skull will occasionally aid in diagnosis, and certain special views are advised.

Many operative approaches have been designed for excision of anterior encephaloceles, and a large number of autogenous and heterogenous substances have been employed for closure of the dural defect incurred by their removal. Water-tight repair of the dura mater is the sole key to successful intervention despite the method through which it is accomplished. Cranioplasty and treatment of the extracerebral mass are of secondary importance.

We prefer the transcranial approach for reasons outlined. Prophylactic antibiotic therapy should be resorted to in every patient, if feasible. Illustrative cases demonstrate various problems encountered in treating these difficult deformities.

A number of articles on this subject were translated from Russian into English for the purpose of this report but owing to lack of space were not included as references. These translations are available at the Mayo Clinic medical library.

Mayo Clinic.

REFERENCES

1. Anderson, F. M.: Intranasal (Sphenopharyngeal) Encephalocele: Report of Case with Intracranial Repair and a Review of Subject, Arch. Otolaryng. 46:644-654 (Nov.) 1947.

2. Mood, G. F.: Congenital Anterior Herniations of Brain, Ann. Otol. Rhin. & Laryng. 47:391-401 (June) 1938.

3. Gisselsson, L.: Intranasal Forms of Encephalomeningoceles, Acta oto-laryng. 35:519-531, 1947.

4. Moore, P. M.: Intranasal Encephalomeningocele: Report of Case, Laryngoscope 62:659-677 (July) 1952.

5. McGillicuddy, O. B.: Encephalomeningoceles in Nasal Cavities, Ann. Otol. Rhin. & Laryng. 51: 516-525 (June) 1942.

6. Schwidde, J. T.: Spina Bifida: Survey of 255 Encephaloceles, Meningoceles, and Myelomeningoceles, A. M. A. Am. J. Dis. Child. 84:35-51 (July) 1952.

7. Ingraham, F. D., and Swan, H.: Spina Bifida and Cranium Bifidum: Survey of 546 Cases, New England J. Med. 228:559-563 (May 6) 1943.

8. Murphy, D. P.: Etiology of Congenital Malformations in Light of Biologic Statistics, Am. J. Obst. & Gynec. 34:890-897 (Nov.) 1937.

9. Sternberg, H.: Zur formalen Genese der vorderen Hirnbrüche (Encephalomeningocele anterior), Wien. med. Wchnschr. 79:462-466 (March 30) 1929.

10. Davis, E. W.: Gliomatous Tumors in Nasal Region, J. Neuropath. & Exper. Neurol. 1:312-319 (July) 1942.

11. Süssenguth, L.: Über Nasengliome, Arch. path. Anat. 195:537-544, 1909.

12. Berger, L., and Coutard, H.: L'esthésioneurocytome olfactif, Bull. Assoc. franç. étude cancer 15:404-414 (Nov.) 1926.

13. Mendeloff, J.: The Olfactory Neuroepithelial Tumors: A Review of the Literature and Report of 6 Additional Cases, Cancer 10:944-956 (Sept.-Oct.) 1957.

14. Lillie, J. C.: Sugar and Chloride in Fluid from Intranasal Cysts, A. M. A. Arch. Otolaryng. 58:166-167 (Aug.) 1953.

15. Richter, A. G.: Medicinische und chirurgische Bemerkungen, vorzüglich im öffentlichen akademischen Hospitale, Berlin, 1813, Vol. 2, pp. 76-81.

16. Fenger, C.: Basal Hernias of the Brain, Am. J. M. Sc. 109:1-17 (Jan.) 1895.

17. Volynkin, N. M.: Surgical Treatment of Frontal Cerebral Hernias, Vopr. neyrokhir. 20: 41-42, 1956.

18. Kovalenka, P. P.: Use of Preserved Cartilage in Surgical Treatment of Frontal Cerebral Hernias in Early Childhood, Vopr. neyrokhir. 20:43-44, 1956.

Surgical Treatment of Thrombotic Occlusion of Aorta and Iliac Arteries

The Leriche Syndrome

JOHN M. ERSKINE, M.D.; FRANK L. GERBODE, M.D.; S. W. FRENCH III, M.D., and R. MAURICE HOOD, M.D., San Francisco

Introduction

It was not until 1923 that Leriche [1] first described the syndrome resulting from the arteriosclerotic obliteration of the lower abdominal aorta, and in 1946 dos Santos [2] succeeded in the first direct surgical attack on the obliterative process of the distal aorta through a thromboendarterectomy. Because the surgical methods of handling this condition are still in a developmental stage, it was considered of value to summarize our series of 33 cases treated surgically during the period from 1953 through 1957.

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From the Department of Surgery, Stanford University School of Medicine, San Francisco; Letterman Army Hospital, San Francisco, and U. S. Naval Hospital, Oakland, Calif.

Clinical Findings

Complete or incomplete occlusion of the distal aorta and the common iliac arteries produces a somewhat varied group of symptoms. The onset is usually gradual, the first symptoms appearing in most of these patients in the fifth decade of life and in a few in the fourth and sixth decades, but in none after 60 years of age (Fig. 1). It is therefore a condition appearing in the middle years of life, and in most instances there is a gradually progressive course. The duration of symptoms before surgery was rather variable, though

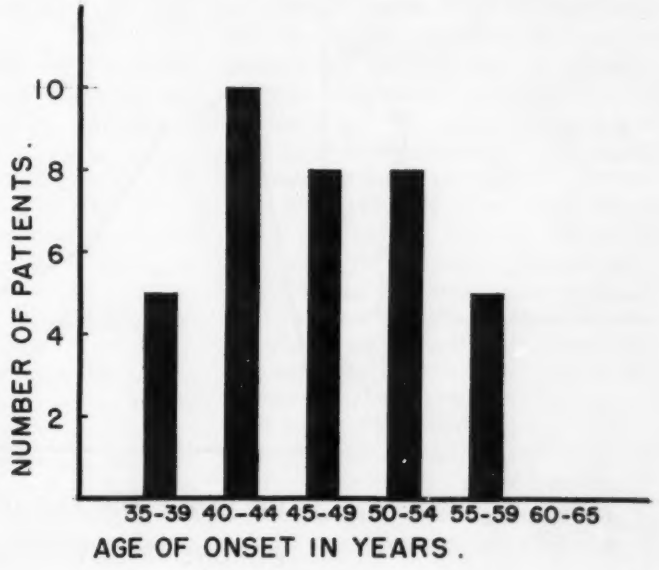

Fig. 1.—Age at onset of symptoms.

TABLE 1.—*Duration of Symptoms, in Years* (Time from Onset to Operation)

Time, Yr.	No. Patients	Time, Yr.	No. Patients
½	3	6	2
1	1	7	0
1½	1	8	3
2	8	9	2
3	1	10	2
4	2	11 or more	2
5	4		

all had symptoms for at least 6 months and several had symptoms for 10 years or more (Table 1). Seventy-two per cent of patients were men.

As one might expect, the symptoms common to all these patients was intermittent claudication, but the location of the pain in some persons was more widespread than in others. All had pain in the lower legs, and 94% had pain in the thighs and hips as well. On the other hand, only 48% experienced pain in the lower-back area, and in 6% the pain was unilteral.

All these patients, while walking on the level, noted claudication within three blocks, and 63% noted it within one block. The distance walked before claudication appeared gradually diminished with the time since the onset of symptoms, and at the time they were finally treated surgically 33% could walk only half a block before the onset of pain (Fig. 2).

Another complaint very frequent among the male patients of this group was inability to sustain an erection. All those in whom this point was specifically checked had noted this defect. Coldness of the feet was noted by many, but not by all, of the group; 73% experienced this, whereas the remaining 27% did not complain of a temperature change in their feet. Cramps in the legs were mentioned by 42%, and 27% noted a form of rest pain as part of the symptom complex.

The preoperative evaluation of this group of patients by the usual history and physical and laboratory examinations revealed that the incidence of clinically detectable disease other than arteriosclerotic disease was relatively low. Three had evidence of cardiac disease, two of renal disease, and two of hypertension. There were no diabetics in this group.

As one might expect, in the majority of cases the femoral pulsations were not palpa-

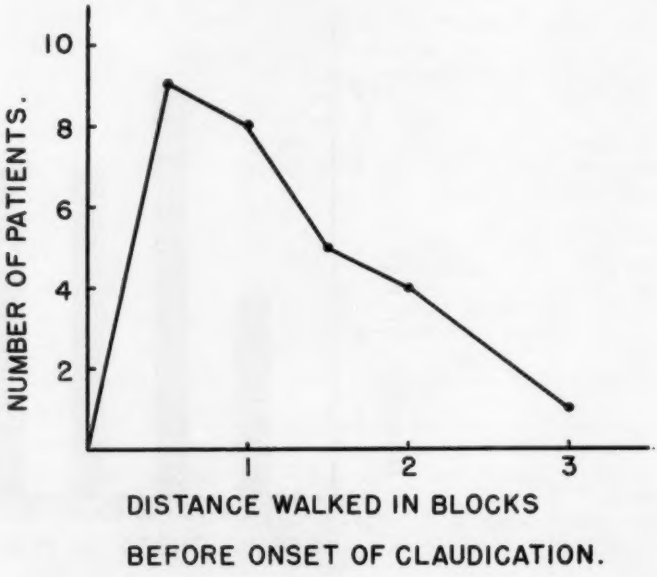

Fig. 2.—Distance walked, in blocks, before onset of claudication.

ble, but in a third of the cases weak femoral pulses were felt. In some instances the abdominal aortic pulsation was present, while in other cases a pulse in that area could not be felt. The pedal and popliteal pulses were not present except in the two cases where the obstructive process involved only the contralateral common iliac artery.

Wasting of the legs was not nearly as prominent when the block was in the aortic-iliac area as when it was in the vessels just above or below the knee. Atrophic changes were noted in only 30% of the patients, and in all instances they were mild. Some decrease in skin temperature was noted in about 60% of the cases; in the other 40% the temperature was considered to be normal. Mild to moderate pallor was noted in most of the cases.

Calcification of the abdominal aorta and its branches was present in 75% of the patients x-rayed. Intravenous pyelograms were normal in every case in which they were taken. Aortograms were made in only 58% of this group, and, although they were an addition from the diagnostic point of view when done, their absence in the cases in which they were not obtained did not seem to alter the plan or success of the subsequent surgical procedure.

Operative Findings and Complications

The upper limit of the obstruction was at the renal vessels in 3%, immediately below the renal vessels in 45%, around the level of the inferior mesenteric vessel in 38%, at the bifurcation of the aorta in 9%, and in the proximal portion of one or both common iliac arteries in 5% of the cases. Thus, in the great majority of cases the major area of involvement was the lower abdominal aorta.

The lower limit of the occlusive process was more uniform in that it extended down to the distal common iliac artery in 80% of the cases. In most instances both common iliacs were obstructed, although in some the occlusion was unilateral (Fig. 3). In 6% the block ended in the lower aorta,

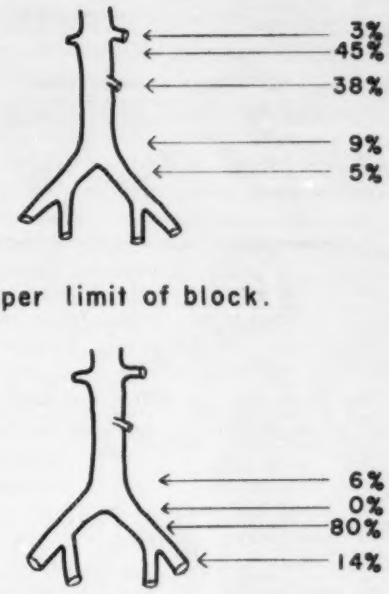

Fig. 3.—Diagrammatic representation of the aortic-iliac area showing, in the upper diagram, the proximal level of the arteriosclerotic occlusion and, in the lower diagram, the distal level of the occlusive disease, expressed in percentages.

and in the remaining 14% it extended into the external iliac arteries. The obstruction was described as complete in 60% of the group, and in 40% the lumen, though markedly narrowed, was not completely occluded.

Resection of the occluded segment with grafting of the defect was the usual method of dealing with the problem and was carried out in 82% of these cases. Homografts were used in 23 cases, and one or another of the synthetic prostheses in 4 instances. A thromboendarterectomy was used in the other 18% of the cases. On the whole, resection and grafting were carried out when the obstructive process was extensive, and thromboendarterectomy was used on the more distal and limited aorto-iliac occlusions. Lumbar sympathectomy was performed in 60% of these patients.

Complications, once they start developing in a major surgical case, are often multiple,

TABLE 2.—*Circulatory Changes—Percentages* of Palpable Pulses in Early Postoperative Period*

Vessel	Strongly Palpable		Weakly Palpable		Absent	
	Bilaterally	Unilaterally	Bilaterally	Unilaterally	Bilaterally	Unilaterally
Femoral	82%	9%	6%	6%	3%	3%
Popliteal	73%	7%	0	3%	20%	3%
Pedal (One or both)	75%	6%	0	3%	19%	3%

* Percentages figured on the 32 patients who survived the operation and early postoperative period.

and in this series most of the complications were confined to two people. In one patient hemorrhage and thrombosis developed at one of the iliac anastomoses, requiring reoperation and regrafting, and finally resulted in gangrene of the distal end of his right third toe. The other instance of thrombosis resulted in our only postoperative death, and occurred in a man whose occlusive process extended from the renal artery down to the distal external iliac, and the postoperative thrombosis led to infarction of the kidneys, uremia, wound disruption, and, finally, death with a cerebral vascular accident. One man developed chronic uremia in the postoperative period, which was apparently precipitated by the operative procedure and responded only very slowly to prolonged hospital care. He died 14 months later, of serum hepatitis, after having received a blood transfusion for the anemia secondary to the uremia. There were two wound infections, and two patients developed a hernia in the incision. There was one cardiac complication in the postoperative period. In addition to the two deaths already mentioned, there were three other deaths in the follow-up period, but they were unrelated to the surgery or the aortic-iliac disease. Thus, the operative mortality was 3%, and 15% of the entire group is now dead.

Results

The percentages of strongly and weakly palpable pulses in the postoperative period and in the later follow-up period are listed in Tables 2 and 3. Eighty-two per cent had strongly palpable femoral pulses bilaterally in the early postoperative period, and another 6% had weakly palpable pulses. Months or years later, of the patients surviving through the follow-up period, there were still 80% with strongly palpable femoral pulses, and in 12% weak femoral pulses were felt bilaterally. There were instances, as can be seen from the Tables, in which the pulse was strongly palpable on one side and only weakly palpable or absence on the other side. Naturally, the percentage of palpable pulses diminished somewhat in the more distal pulses.

Of course, the percentage of palpable pulses does not tell the whole story with regard to the degree of rehabilitation of these patients, for 93% were considered to have some improvement in the temperature of the feet. The presence or absence and/or the degree of change of the intermittent claudication is also important, though more

TABLE 3.—*Circulatory Changes—Percentages* of Palpable Pulses in Later Follow-Up Period*

Vessel	Strongly Palpable		Weakly Palpable		Absent	
	Bilaterally	Unilaterally	Bilaterally	Unilaterally	Bilaterally	Unilaterally
Femoral	80%	0	12%	4%	4%	4%
Popliteal	60%	4%	8%	0	28%	4%
Pedal	56%	4%	4%	4%	32%	8%

* Percentages figured on the 25 patients alive and available for this follow-up study.

subjective, gauge. Of the 27 patients in the follow-up period responding to this question, 37% had no claudication; 33% had some, but only after walking a considerably greater distance; 26% had some, with its onset at a slightly greater distance than before operation, and 5% noted no change in this symptom following the procedure. Of the surviving patients, 60% were doing full-time work; 8% were engaged in part-time work; 16% did housework only, and 16% stated they did not work. There were no instances of major progression of the vascular disease in the aorta or distal vessels in this follow-up period. Of those rating the results of the operation, 76% stated that they were markedly improved; 16% thought there was moderate improvement; 4% noted only slight improvement, and another 4% considered themselves unchanged.

Comment

The lower limit of the complete or incomplete block in these patients with aortic-iliac disease was in the distal common iliac artery in 80% of the cases, and apparently this was a significant area in the origin of this particular manifestation of arteriosclerosis. The process seemed to start most frequently just proximal to the bifurcation of the common iliac arteries as arteriosclerotic calcific changes of the intima and media and associated chronic inflammatory reaction of the adventitia. It progressed largely in the proximal direction into the area of the aortic bifurcation and above; and, as the lumen of the artery progressively narrowed, a point was reached where thrombosis occurred, with formation of a coagulum, which eventually became organized to a certain degree. A fairly clear-cut pattern of arteriosclerotic obliterative disease is thus recognized, and in most of these cases certain of the other patterns, such as extensive involvement of the superficial femoral arteries or occlusions of the arteries beyond the popliteal vessels, were not present. This was very fortunate, for when a satisfactory flow of blood was established between the proximal abdominal aorta and the bifurcation of the common iliac arteries, definite improvement was obtained in the great majority of patients.

In addition, once the circulation was satisfactorily reestablished, the improvement remained over the follow-up period of one to four years, with no instances of reocclusion of the aortic-iliac area and relatively infrequent evidence of active progression of the arteriosclerotic process in the femoral or more distal arteries. In this series there were no instances of gangrene developing during the follow-up period. In addition, the deaths in the follow-up period were primarily cardiac or renal rather than from a progression of the disease in the large vessels.

The question of which surgical procedure should be employed for the relief of the vascular obstruction is of importance. Certainly there is no one way of handling this technical problem, and good results are obtained by three different methods. Freeman [3] and Wylie [4] have emphasized the use of thromboendarterectomy in the majority of their cases, with very satisfactory results in most cases. Welch [5] and Julian [6] have both emphasized resection of the occluded segment and grafting of the defect. De Bakey [7,8] makes use of both these methods, but he also uses the by-pass technique, in which the diseased segment is left in place and the graft conducts blood around the obstruction. As can be seen, we have emphasized the use of resection and grafting in the great majority of our cases, but we have used the thromboendarterectomy in a few of the cases with limited obstructions and little or none of the inflammatory element in the vessel wall. We have not had occasion to use the by-pass method. It is our feeling that when the obliterative process involves a rather long segment of vessel, resection and grafting is associated with less morbidity and mortality and is easier technically. The incidence of immediate or recurrent thrombosis also seems to be less in the grafted group than in patients treated entirely by endarterectomy. Freeman [3] re-

cently reported his experiences in 87 cases of limited or extensive aortic-iliac occlusions treated by thromboendarterectomy, and there was an operative mortality of 8%, immediate failure in 11%, recurrent thrombosis in another 14%, and hemorrhage which did not result in death in another 8%. In our series of six cases in which the obstruction was limited in extent and thromboendarterectomy was employed, there were no deaths, no immediate failure, and no postoperative hemorrhages, and recurrent thrombosis developed on one side in one case only. In the 27 cases with more extensive disease where grafting was employed, the results were also somewhat more satisfactory than in a group with more extensive disease treated by thromboendarterectomy, in that there was an operative mortality of 3%, an immediate failure rate of 10%, and no late thromboses. There was one instance of postoperative hemorrhage (3%).

De Bakey [7,8] also apparently limits the use of thromboendarterectomy to well-localized occlusive processes with minimal medial and adventitial mural involvement (one-fourth of his cases), and he reports successful results in 95% of these cases, with a 2.8% mortality. These figures are almost identical with the results he obtained in the 31% of the cases treated by resection and grafting where the vessel changes were extensive and destructive, but still well localized to the aortic-iliac area. In addition, in almost 50% of his cases he used excision with graft replacement and by-pass, or by-pass alone, with highly satisfactory results and minimal mortality. It is extremely difficult to compare figures from different clinics because their initial and late evaluation of the cases may be based on different criteria. Certainly the question of the type of surgical therapy to be employed is still in the development stage, and for the present at least the surgeon should be familiar with several methods and fit the method to the particular situation encountered.

The importance of aortography in the evaluation of these patients for surgery is another somewhat controversial matter. Aortograms were used in 58% of our patients. In the cases in which it was not done no complications or unusual difficulties were encountered that might have been avoided had an arteriogram been made. The knowledge gained by doing it did not seem to alter the therapeutic approach to the problem. The one operative mortality had had an arteriogram; so the complications and ultimate death in that case could not be attributed to lack of study. One patient not listed in this series because he was not operated upon developed permanent paraplegia after aortography. Other complications of and deaths from aortography have been reported.

We have had the opportunity to study a homograft removed from a patient in this

Fig. 4.—Photograph of an arterial homograft in the distal abdominal aorta which had been in situ for two years before the patient died of cardiac disease. Note the extensive arteriosclerotic disease both above and below the graft and the smooth appearance of the graft itself, which shows no evidence of atheromatous or aneurysmal changes.

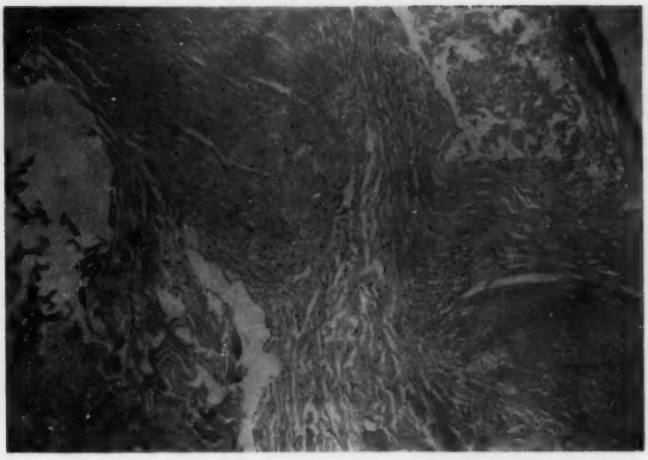

Fig. 5.—Junction of the artery and the homograft. Note the graft to the right and the artery above and to the left. Suture material is on the far left, and the lumen of the vessel is at the upper right-hand corner, with endothelium crossing the scar-tissue union of the graft and the artery. Hematoxylin-eosin stain.

group who died of cardiac failure two years after the grafting procedure. Grossly, as can be seen from the illustration (Fig. 4), the graft in the lower abdominal aorta looked far better than the aorta above or below the graft. Photomicrographs of the suture lines between the aorta and the homograft and of the midsection of the graft are also shown (Figs. 5, 6, 7, and 8), and the graft wall, though thinned out, has not developed arteriosclerotic changes. No aneurysmal changes were present.

Summary

The 33 cases with aortic-iliac occlusions treated surgically between 1953 and 1957 have been analyzed with regard to history, physical findings, surgical procedures used, and immediate and late results.

The clinical symptoms and physical findings in the group of patients are on the whole similar to those described by Leriche and others. Typically, the symptoms appear most frequently in men in the fifth decade of life, with gradual progression of inter-

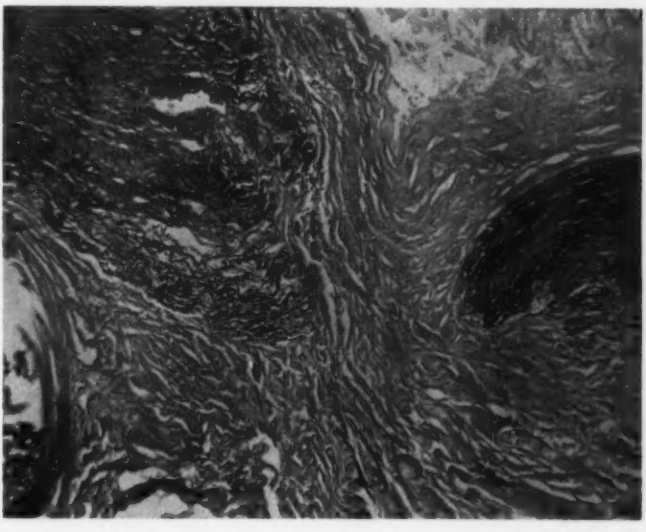

Fig. 6.—Elastic-tissue stain of section similar to that shown in Figure 5. Note the compact elastic tissue in the graft, as shown on the right, with loss of smooth-muscle elements of the media.

Fig. 7.—Elastic-tissue stain of the wall of the homograft, showing the thinned-out graft, composed mostly of the elastic-tissue elements of the media, supported by a heavy layer of scar tissue.

mittent claudication, which is located in the hips and thighs, as well as the lower legs. In addition, there is usually an inability to sustain an erection, and sometimes coldness of the feet. The femoral pulses are absent or very weak, and the more distal pulses are absent. There is little or no atrophy or color change in the lower legs and feet, and the feet may not even feel particularly cold.

Calcification is usually found to be present in the abdominal vessels by x-rays; aortograms do not seem to be essential in making the diagnosis or in planning the therapy, but may be required in special circumstances.

In this series the block was complete in most cases and often extended from just below the renal vessels to just above the bifurcation of the common iliac arteries. If

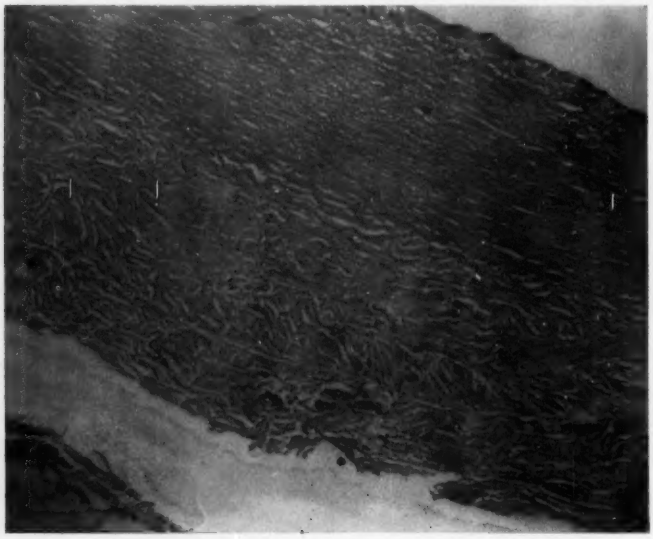

Fig. 8.—Section of the homograft wall with the lumen at the top right. Hematoxylin-eosin stain.

the block was limited in extent, a thromboendarterectomy was sometimes carried out; but in the majority of cases a resection with grafting was the procedure performed. A bilateral sympathectomy was usually added. The operative mortality was 3%, and the results were highly satisfactory through the follow-up period in over 90% of the patients, from both the objective and the subjective point of view.

Stanford University Hospitals, Clay and Webster Sts.

REFERENCES

1. Leriche, R.: Des oblitérations artérielles hautes (oblitération de la terminaison de l'aorte) comme causes des insuffisances circulatoires des membres inférieurs, Bull. et mém. Soc. Chir., Paris 49:1404, 1923.

2. dos Santos, J. C.: Sur la désobstruction des thromboses artérielles anciennes, Mém. Acad. chir. 73:409, 1947.

3. Freeman, N. E., and Nicholson, G. J.: Evaluation of Thromboendarterectomy in Treatment of Obliterative Arterial Disease, A. M. A. Arch. Surg. 78:280, 1959.

4. Wylie, E. J.: Thromboendarterectomy for Arteriosclerotic Thrombosis of Major Arteries, Surgery 32:275, 1952.

5. Welch, J. S.; Kirklin, J. W.; Ellis, F. H., Jr., and Bruwer, A. J.: Resection and Grafting for Chronic Occlusion of the Terminal Aorta or Iliac Arteries, J. A. M. A. 164:1045, 1957.

6. Julian, O. C.; Grove, W. J.; Dye, W. S.; Olwin, J. S., and Sadove, M. S.: Direct Surgery of Arteriosclerosis: Resection of Abdominal Aorta with Homologous Aortic Graft Replacement, Ann. Surg. 138:387, 1953.

7. De Bakey, M. E.; Creech, O., Jr., and Cooley, D. A.: Occlusive Disease of the Aorta and Its Treatment by Resection and Homograft Replacement, Ann. Surg. 140:290, 1954.

8. De Bakey, M. E.; Crawford, E. S.; Cooley, D. A., and Morris, G. C., Jr.: Surgical Considerations of Occlusive Disease of the Abdominal Aorta and Iliac and Femoral Arteries: Analysis of 803 Cases, Ann. Surg. 148:306, 1958.

9. Halpert, B.; Erickson, E. E.; De Bakey, M. E.; Creech, O., Jr., and Cooley, D. A.: Occlusive Disease of the Abdominal Aorta: Structural Alterations, A. M. A. Arch. Path. 65:158, 1958.

Treatment of Squamous-Cell Carcinoma of the Floor of the Mouth

JOHN B. ERICH, M.D., and LYLE V. KRAGH, M.D., Rochester, Minn.

Before 1920, few operations intended to cure carcinoma of the floor of the mouth were proposed. Because of the morbidity and the high mortality rates involved in radical surgical removal of malignant lesions in this location, early surgeons were led to search for more effective but less dangerous methods. Irradiation with radium and by external sources was being perfected and was looked on with high hope. After a thorough trial period, it became clear that the primary tumor often could be controlled by irradiation but that metastatic lesions of the cervical lymph nodes seldom were cured by irradiation alone. At some institutions, electrosurgical removal of the primary lesion was undertaken with some degree of success. Thus, for many years, the usual treatment for malignant tumors in the floor of the mouth consisted in eradicating the primary lesion by electrocoagulation or irradiation or both, and in treating lesions in the cervical lymph nodes by some type of block dissection.

With the more recent surgical advances (antibiotics, better anesthesia, blood replacement, and better preoperative and postoperative nutrition), radical open surgical removal of these primary oral lesions has been attempted with success. This has led to removal of the primary lesion and the cervical lymphatic structures at the same time. More recently, such techniques have been extended to en bloc removal of the primary lesion, the cervical lymphatic structures, and the intervening tissue. Only a few reports on the survival of patients treated by these methods have appeared in the literature as yet. As might be expected, the reports are based on selected groups of cases. Since these surgical procedures probably cannot be applied to all malignant growths in the floor of the mouth, accurate data regarding the effectiveness of these operations probably will not be forthcoming in the very near future.

It is the purpose of this study to determine the end-results of treatment of all patients who had squamous-cell carcinoma of the floor of the mouth seen at the Mayo Clinic, from 1945 through 1954, and, if possible, to determine the efficacy of the various methods of treatment.

General Comment on Treatment Used

During this 10-year period, patients with squamous-cell carcinoma of the floor of the mouth were managed on an individual basis, the treatment not being restricted to any one method. During the early years of the study, if the primary lesion was considered removable, it was usually extirpated by electrosurgery. If the lesion was small and well localized, it usually was excised with a margin of normal surrounding tissue. In some of the cases in which the primary lesion was treated by electrocoagulation, block dissection of the neck and ligation of the external carotid artery preceded treatment of the oral lesion. In some instances, depending on the extent of the lesion, the histologic grade of the tumor, or a question of incomplete removal of the lesion, supplementary interstitial radium or radon seeds were used. More extensive, high-grade, less localized lesions usually were treated by

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Section of Plastic Surgery, Mayo Clinic and Mayo Foundation (Dr. Erich). Fellow in Plastic Surgery, Mayo Foundation (Dr. Kragh). The Mayo Foundation, Rochester, Minn., is a part of the Graduate School of the University of Minnesota.

radium or radon seeds, or roentgen therapy, or both. During the later years of the study, composite operations occasionally were employed.

Treatment of the cervical lymphatic structures likewise was individualized. In general, prophylactic dissection of the neck has been employed at the Clinic in the treatment of occult cervical metastatic tumors from carcinoma of the floor of the mouth. However, factors such as age, general health, and the extent and histologic grade of the lesion were considered in its application. Some patients refused such prophylactic dissection. During the early years of the study, dissection of the upper part of the neck (submental, submaxillary, and upper deep-jugular lymph nodes) frequently was employed, often bilaterally, since many of the lesions were near or crossed the midline. Also, block dissection of the neck, saving the internal jugular vein or the sternocleidomastoid muscle or both, occasionally was employed. During the later years of the study, the standard radical block dissection of the neck was employed more frequently. More recently, this technique occasionally has been employed as part of a composite operation. Prophylactic roentgen therapy to the neck was used occasionally during the early years of this study only. Cervical metastatic lesions, if they were thought to be inoperable, or if they occurred in aged, poor-risk patients, usually were treated by use of interstitial radon seeds or external irradiation or both.

Factors of Selection

During the 10-year period of this study, 102 patients with histologically proved invasive squamous-cell carcinoma of the floor of the mouth were treated at the Clinic.* This group of 102 patients forms the basis for this study.

All patients who had proved squamous-cell carcinoma of the floor of the mouth and who received any form of treatment at the Clinic are included, regardless of how short was the period of observation. Patients who had primary malignant tumors of the floor of the mouth other than squamous-cell carcinoma were excluded. Nine patients treated at the Clinic were excluded because inadequate histologic evidence was present in the record to allow inclusion in the study. Patients with in situ carcinoma (five cases) were excluded. A total of 33 patients who did not receive any treatment here were excluded. These patients came for consultation only, refused treatment, sought treatment elsewhere, or were hopelessly incurable and returned home for palliative treatment.

Data in Present Study

Age and Sex Distribution.—The mean age of the 102 treated patients was 57.2 years, with an age range of 38 to 80 years; 93 patients were men, and 9 were women.

Location and Extension of Lesions.—A total of 34 tumors arose on the right side, and 36 arose on the left; 24 originated in the midline of the floor of the mouth. The exact location of the tumor was not stated in the remaining eight instances.

Extension of the tumor to the tongue was present on admission in 20 patients, and extension to the mandible was present in another 20. Extension of a fairly well-lateralized tumor across the midline of the floor of the mouth was present in 16 patients. Clinically involved cervical lymph nodes were present in 38 instances.

Previous Treatment.—Of these 102 patients, 22 had received some treatment in an attempt to control the tumor prior to admission to the Clinic (excluding biopsy, dental treatments, and local applications).

Histologic Grade of Lesion.—The degree of malignancy of the tumors (classification of Broders) was recorded. Two tumors were Grade 1; 60 were Grade 2; 34 were Grade 3, and 6 were Grade 4.

* One may have some difficulty in defining the nebulous zone called the "floor of the mouth." For our purposes, it is defined as that region between the lateral border of the tongue and the mandibular alveolus. During this same 10-year period, 269 patients with squamous-cell carcinoma of the anterior portion of the tongue and 118 patients with squamous-cell carcinoma of the mandibular gingiva were treated at the Clinic. Studies on these patients are being published separately.[5,6]

TABLE 1.—*Squamous-Cell Carcinoma of the Floor of the Mouth: Initial Treatment of the Primary Lesion at the Mayo Clinic*

Treatment	Patients
Excision (includes excision as part of a composite operation)	32
Electrocoagulation	36
Excision or electrocoagulation plus radium or radon	18
Radium or radon, or external irradiation, or both	13
No treatment	3
Primary controlled	2
Died before treatment	1
Total	102

Multiple Cancers.—Additional malignant tumors were present, or developed later, in 18 of the 102 patients; in 9 instances these additional tumors were in the oral cavity.

Treatment of Primary Lesion.—Table 1 indicates the initial treatment instituted at the Clinic for control of the primary lesion of the floor of the mouth. To prevent confusion, only the first attempt at control of the primary lesion is listed. Obviously, the first therapeutic attempt is the most important in the ultimate control of the lesion. It must be appreciated, however, that further measures were employed in those cases in which the primary lesion was not controlled by the initial therapy.

Although many patients returned to the Clinic for follow-up examinations, many others did not. When the follow-up was inadequate, it was impossible to determine whether the primary lesion was controlled or not. In some cases, we were unable to determine whether death was due to recurrence of the malignant tumor or to other causes. Also, in those patients known to have died of carcinoma, information often was lacking as to whether the primary lesion was controlled. Therefore, an accurate estimate of the efficacy of control of the primary lesion could not be made from this study.

Treatment of Cervical Lymphatic Structures.—In 67 cases block dissection of the cervical lymphatic structures was the initial treatment to the neck; this includes those dissections done as part of a composite operation. Radon or roentgen therapy to the lesions in the neck was used as a primary measure in 12 cases. As was previously stated with regard to the treatment of the primary oral lesions, these measures represent only the first attempt at control of the cervical metastatic lesions. If the metastatic tumors were not controlled by these measures, further therapy obviously was employed in an attempt to eradicate these metastatic tumors. A total of 23 patients did not receive any treatment to the neck at any time during the course of this study.

For the same reasons stated previously regarding control of the primary lesions, it was impossible to ascertain with any accuracy the efficacy of control of these metastatic lesions in the neck.

Incidence of Cervical Metastasis.—Some form of block dissection of the cervical lymph nodes was done 68 times when involved lymph nodes were clinically absent; this number includes bilateral block dissections, which were counted as two procedures. Histologically, nodes were found to be involved by carcinoma in 6 of these 68 dissections (9%). Likewise, a total of 48 block dissections were done in cases in which lymph nodes appeared clinically to be involved. Histologically, nodes were found to be involved in 31 of these operations and to be uninvolved in 17, giving an "error rate" of 35%.

Crossed Metastasis.—In 30 patients histologic proof of cervical metastasis was available as a result of excision of nodes or block dissection of the neck. Of these 30 patients, 16 had well-lateralized lesions, and 14 had lesions in the anterior of the midline aspect of the floor of the mouth. Of the 16 tumors that were well lateralized, 10 metastasized to the homolateral nodes, 3 spread to the contralateral cervical nodes, and 3 spread bilaterally. Of these last three lesions, one spread to the homolateral side first, and the other two spread bilaterally at about the same time. Of the 14 poorly lateralized tumors, 9 metastasized to one side of the neck, and 5 spread bilaterally.

TABLE 2.—*Squamous-Cell Carcinoma of the Floor of the Mouth: Survival Rates*

Period, Yr.	Patients		Lived Beyond Indicated Period	
	Total	Traced	Number	Per Cent *
3	102	94	57	60.6
5	73	65	30	46.2

* Based on traced patients; inquiry as of Jan. 1, 1958. Included in the three-year group are only those patients treated three or more years prior to time of inquiry, that is, in 1954 or earlier. The five-year group includes only those treated in 1952 or earlier. Hospital deaths are excluded in the calculation of the survival rates.

Survival of Patients

Table 2 summarizes the survival data for these 102 patients. The five-year survival rate is 46%, and the three-year survival rate is 61%.

The survival of patients according to the method of treatment employed was studied, an effort being made to compare the survival with the extent and severity of the presenting lesion. As would be expected, difficulties arose in estimating the extent of the lesion from the description given in the records. Considering both the extent of the primary lesion and the presence of cervical metastatic tumors, no significant differences in the survival rates according to the method of treatment employed could be found. Patients who had small, well-localized lesions that were treated by electrocoagulation or excision of the primary lesion, the neck never becoming involved, had a relatively good survival rate. Although a fair number of patients were treated by radium or radon seeds, or external irradiation, or both, the severity of these lesions was estimated to be about the same as that of the entire group of tumors, and the survival of these patients was also about the same. The large intermediate group of patients treated by excision or electrocoagulation of the primary lesion and block dissection of the cervical lymphatic structures differed little in the five-year survival rates. The survival rates for the groups having combined and composite operations were somewhat poorer; however, the pa-

TABLE 3.—*Carcinoma of the Floor of the Mouth: Reported Series*

Authors	Years of Treatment	No. of Cases	Factors of Selection	Predominant Treatment		Survival Rates		
				Primary Lesion	Neck	5-Year Determinate	5-Year Absolute	3-Year
Martin and Sugarbaker	1930-1934	103	Unselected	Irradiation	Irradiation	22/87=25%	22/103=21%	—
Cade	1925-1951	118 total 97 eligible	Unselected	Irradiation	Irradiation and dissection	24/89=27%	24/97=25%	—
Gardham	1920-1947	72	Presumably unselected	Irradiation and surgery	Irradiation and dissection	—	20/72=28%	—
Windeyer	1931-1946	131 total 115 treated	Floor of mouth and mandibular alveolus	Irradiation	Dissection	33/115=29%	—	—
Dobbie	1932-1945	427	Presumably unselected	Irradiation	Dissection	—	32%	—
Mattick and associates	1937-1945	98 total 82 determinate	Unselected	Irradiation	Irradiation	18%	15%	—
Ash and Millar	1929-1949	95 eligible	Presumably unselected	Irradiation	Dissection	43%	39%	—
Alford and Klopp	—	20 total 11 eligible	Localized to floor, with or without nodes	Surgery	Dissection	—	7/11	15/20=75%
Present study	1945-1954	102 total 73 eligible	All patients treated	Surgery or electrocoag.	Dissection	46%	—	61%

tients treated by these methods appeared to have somewhat more extensive lesions.

Because only a relatively small number of cases were present in each category of therapeutic method and because of the many variables encountered in this study, exacting conclusions could not be reached regarding the most efficacious method of treatment of squamous-cell carcinoma of the floor of the mouth. Of most significance is the five-year survival rate of 46.2% of the traced patients. Table 3 lists some of the survival rates in other reported series of carcinoma of the floor of the mouth. In many instances, these lesions are included with lesions of the tongue or the gingiva. This grouping must be taken into consideration in comparing survival data.

Comment

As noted earlier, the past decade has seen an increasing employment of extended radical and composite operations for intraoral cancers. Although most authors are enthusiastic about these methods, most of these extensive operations have been done only recently, and survival data on large series of patients treated by these methods are not yet available. The few available reports usually represent small series of selected patients. As was emphasized by Morfit, only when large series of unselected patients treated by certain methods are used for comparison can the true value of the procedures be evaluated.

As will be noted in Table 3, although the patients were treated by different methods, little difference is present in the survival rate among the reported series. During the period covered by our present study, the great majority of the patients were treated by excision or electrosurgical removal of the primary lesion and by some type of block dissection of the cervical lymphatic structures. In many of these instances, block dissection of the upper part of the neck was done bilaterally. In many of the patients treated by electrocoagulation, block dissection of the cervical lymphatic structures and ligation of the external carotid artery preceded treatment of the primary lesion. Only a relatively few composite operations were employed during this period. In some of the cases, interstitial radium or radon seeds were used in addition to surgical removal of the primary lesion.

The relatively low incidence (9%) of metastatically involved cervical lymph nodes found in specimens dissected from the neck in the absence of clinical evidence of metastasis surprised us. Also somewhat surprising was the high rate of error (35%) in estimating metastatic spread clinically. These data open the question of the advisability of doing prophylactic dissection of nodes for these lesions. Since many of these tumors arise in or near the midline of the floor of the mouth or cross the midline, the problem of elective dissection of nodes is compounded. Among the patients treated by Alford and Klopp by en bloc removal of the floor of the mouth and bilateral dissection of the upper part of the neck, 20 of 28 had no evidence of involvement of the nodes in the resected specimen. In those who did have metastatically involved nodes, the side of the neck involved was not stated.

With the low incidence of metastatically involved nodes in specimens removed during prophylactic dissection of the neck, plus the large number of the lesions that are essentially in the midline, and in which the pathway of metastasis is less predictable, one must question the advisability of doing prophylactic dissection in these patients. Perhaps radical removal of the primary lesion with observation of the lateral cervical region for appearance of metastasis would be as effective as a composite operation or prophylactic dissection of the neck after treatment of the primary lesion.

Summary

A study was made of 102 patients with squamous-cell carcinoma of the floor of the mouth treated at the Mayo Clinic from 1945 through 1954. All patients who received any form of treatment were included.

An attempt was made to determine the most effective method of treatment of these lesions. Comparison of the survival rates of the patients with the extent of the local and metastatic lesions disclosed no significant difference among the various groups classified according to method of treatment. The patients who had combined treatment of the primary lesion and the cervical lymphatic structures at the same time or who were treated by a composite operation had slightly poorer survival rates, but these patients appeared to have somewhat more extensive lesions.

A five-year survival of 46% of the traced patients was obtained.

Mayo Clinic.

REFERENCES

1. Alford, T. C., and Klopp, C. T.: The Surgical Treatment of Cancer of the Floor of the Mouth, Cancer 11:1-3 (Jan.-Feb.) 1958.

2. Ash, C. L., and Millar, O. B.: Radiotherapy of Cancer of the Tongue and Floor of the Mouth, Am. J. Roentgenol. 73:611-619 (April) 1955.

3. Cade, S.: Carcinoma of the Floor of the Mouth, Brit. J. Surg. 41:225-230 (Nov.) 1953.

4. Dobbie, J. L.: Carcinoma of the Floor of the Mouth, Brit. J. Surg. 41:250-253 (Nov.) 1953.

5. Erich, J. B., and Kragh, L. V.: Results of Treatment of Squamous Cell Carcinoma of the Anterior Part of the Tongue, unpublished data.

6. Erich, J. B., and Kragh, L. V.: Results of Treatment of Squamous Cell Carcinoma Arising in the Mandibular Gingiva, unpublished data.

7. Gardham, A. J.: Carcinoma of the Floor of the Mouth, Brit. J. Surg. 41:241-250 (Nov.) 1953.

8. Martin, H. E., and Sugarbaker, E. L.: Cancer of the Floor of the Mouth, Surg. Gynec. & Obst. 71:347-359 (Sept.) 1940.

9. Mattick, W. L.; Meehan, D. J., and Haberlin, J. P.: Squamous-Cell Carcinoma of the Floor of the Mouth, Surgery 31:575-582 (April) 1952.

10. Morfit, H. M.: Neck Dissection in Treatment of Cancer of the Tongue and Mouth, Current Surgical Management: A Book of Alternative Viewpoints on Controversial Surgical Problems, edited by J. H. Mulholland, E. H. Ellison, and S. R. Friesen, Philadelphia, W. B. Saunders Company, 1957, pp. 387-392.

11. Windeyer, B. W.: Carcinoma of the Floor of the Mouth, Brit. J. Surg. 41:231-237 (Nov.) 1953.

Results of Treatment of Squamous-Cell Carcinoma Arising in Mandibular Gingiva

JOHN B. ERICH, M.D., and LYLE V. KRAGH, M.D., Rochester, Minn.

In other communications,[1,2] we have briefly reviewed the progress of treatment of squamous-cell carcinoma of the anterior portion of the tongue and the floor of the mouth. The treatment of carcinoma of the mandibular gingiva, like that of the two types just mentioned, has passed through many different phases. Prior to this century, the occasional open surgical resection of such lesions was truly a major undertaking. As methods of electrosurgery and irradiation improved, the majority of such lesions were treated by these techniques, and the immediate mortality rate was reduced considerably. However, it became apparent that patients who had malignant invasion of the mandible were cured only occasionally by irradiation. Also, irradiation was found to be unsatisfactory as a treatment of metastatic tumors in the cervical lymph nodes. Thus, the primary lesion usually was treated by irradiation or electrocoagulation, and the cervical metastatic lesions were treated by surgical removal, usually block dissection of the neck.

With the more recent surgical and anesthetic advances, there has been a return to surgical removal of the primary lesion and the involved cervical nodes. More recently, this has been extended to surgical en bloc removal of the mandible and the cervical lymphatic-bearing tissue, the so-called commando procedure. Although these techniques are used fairly extensively now, only a few reports of their efficacy are yet available.

It is the purpose of this study to determine the end-results of the treatment of all patients with squamous-cell carcinoma of the mandibular gingiva treated at the Mayo Clinic from 1945 through 1954, and, if possible, to determine the efficacy of the various methods of treatment.

General Remarks on Treatment Used

During the 10-year period ending in 1954, treatment of patients with squamous-cell carcinoma of the mandibular gingiva at the Mayo Clinic was individualized. During the early years covered by this study, if the primary lesion was thought to be curable, it usually was widely removed by electrocoagulation. Because of the possibility of bleeding following electrocoagulation of the primary lesion, dissection of the neck and ligation of the external carotid artery preceded treatment of the primary lesion in some of these cases. With more extensive local lesions, interstitial radium or radon seeds, or external irradiation, or both, were used, alone or combined with electrocoagulation. During the later years of the study, the same fundamental methods were employed but, in addition, a few partial mandibular resections and in continuity operations were employed.

Prophylactic dissection of the neck has been employed in the treatment of occult metastatic tumors in cervical nodes arising from carcinoma of the lower gingiva. Each patient was dealt with as an individual problem, and the application of such dissection was less rigid than it was for lesions

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Section of Plastic Surgery (Dr. Erich); Fellow in Plastic Surgery, Mayo Foundation (Dr. Kragh), Mayo Clinic and Mayo Foundation. The Mayo Foundation, Rochester, Minn., is a part of the Graduate School of the University of Minnesota.

of the anterior part of the tongue. The age of the patient and his general health, and the size and histologic grade of malignancy of the primary mandibular lesion were factors considered in employing prophylactic dissection of the neck. During the early years of the study, dissection of the upper part of the neck (submental, submaxillary, and upper deep-jugular nodes) was done in addition to the standard radical block dissection. Dissection of the upper part of the neck usually was done bilaterally if the lesion was near the midline. Also during the early years of the study, block dissection of the cervical nodes without sacrifice of the internal jugular vein or the sternocleidomastoid muscle occasionally was employed. During the later years of the study, the standard radical block dissection of the neck was employed more frequently, and eventually was included as part of a composite operation. Prophylactic roentgen therapy to the neck was used occasionally during the early years of this study only. Inoperable cervical metastatic tumors or cervical metastatic lesions occurring in aged, poor-risk patients usually were treated with interstitial radon seeds or external roentgen therapy or both.

Factors of Selection

During the 10-year period of our study, 118 patients with histologically proved invasive squamous-cell carcinoma of the mandibular gingiva were treated at the clinic.* These patients form the basis for this study.

All patients whose lesions arose in the posterior commissure of the mouth and the retromolar region are included. All patients with histologically proved squamous-cell carcinoma who received any form of treatment at the clinic are included, regardless of how little treatment was given or how short was the period of observation. Patients who had malignant tumors of the mandible or mandibular gingiva other than squamous-cell carcinoma were excluded, as were those who had carcinomas that arose on the buccal mucosa or lip and secondarily extended to the mandible. Although treated at the clinic, two patients had inadequate evidence of malignancy for inclusion in this study; another 53 patients received no form of treatment here and also were excluded. These patients came for consultation only, refused treatment, sought treatment elsewhere, or returned home for palliation for advanced malignant tumors. In this latter group, histologic review of tissue was not available in all instances, and some of these may have represented malignant lesions other than squamous-cell carcinoma.

Data in Present Study

Age and Sex Distribution.—The mean age of the 118 treated patients was 66 years, ranging from 38 to 86 years. A total of 101 (86%) of the patients were men and 17 (14%) were women.

Location and Extension of Lesions.—Of these 118 tumors, 58 (49%) arose on the right side, 58 (49%) on the left and 2 (2%) in the midline. Extension to the floor of the mouth was present in 17 (14%) of the cases, and extension to the cheek or lip was present in 36 (31%). In 24 (20%) of instances, the lesion arose in the region of the posterior commissure or extended to the region of the posterior commissure, tonsil, or soft palate. Clinically involved cervical lymph nodes were present on admission in 35 (30%) of the patients.

Involvement of the Mandible.—Extensive malignant involvement of the mandible, demonstrated roentgenographically, was present in 26 (33%) of the 79 patients in whom roentgenologic studies of the jaw were made; minimal involvement or involvement of the alveolar process only was present in another 30 (38%) of these patients. An essentially normal roentgenographic appearance was present in the remaining 23 patients.

* During this same period, 269 patients with histologically proved squamous-cell carcinoma of the anterior part of the tongue and 102 patients with histologically proved squamous-cell carcinoma of the floor of the mouth were treated here, as we have reported.[1,2]

TABLE 1.—*Squamous-Cell Carcinoma Arising in the Mandibular Gingiva: Initial Treatment of the Primary Lesion at the Mayo Clinic*

Treatment	Patients
Excision (includes excision as part of a composite operation)	23
Electrocoagulation	64
Electrocoagulation or excision plus radium or radon	15
Radium or radon, or roentgen therapy, or both	14
No treatment (primary believed controlled)	2
Total	118

Histologic Grade of Lesion.—The degree of malignancy of these tumors (method of Broders) was recorded; 24 (20%) were Grade 1, 60 (51%) were Grade 2; 31 (26%) were Grade 3, and 3 (3%) were Grade 4.

Previous Treatment.—A total of 27 (23%) of the 118 patients had undergone some attempt to control their malignant lesions prior to coming to the clinic (excluding biopsy, local applications, and dental extractions or treatments).

Multiple Cancers.—Additional malignant tumors were present or developed later in 13 (11%) of the 118 patients; in 9 instances, these tumors arose in other portions of the oral cavity.

Treatment of Primary Lesion.—Table 1 summarizes the initial treatment employed at the clinic for control of the primary lesion. It is believed that the initial therapeutic attempt represents the best opportunity to control the lesion; therefore, to prevent confusion, only the initial treatment to the primary tumor is listed in this table. Obviously, those patients whose tumor recurred were treated further.

Not all of the patients returned to the clinic for follow-up. In many instances, information regarding the course of their disease was incomplete. In some cases, the actual cause of death could not be determined; in many of those who were known to have died of recurrent carcinoma, we were unable to determine whether the primary lesion was controlled. Thus, an accurate over-all estimate of the efficacy of control of the primary lesion could not be made from this study.

Treatment of Cervical Lymphatic Structures.—In 57 patients (48%), block dissection of the cervical lymphatic structures, including those dissections done as part of a composite operation, was done as the initial treatment to the neck. Radon or roentgen therapy to the lesions in the neck was used as an initial therapeutic attempt in 19 (16%) of the patients. It must be remembered that these procedures represent only the first attempt at control of the lesions in the neck; obviously, if the metastatic lesions were not controlled by these methods, other treatment was employed. The remaining 42 patients (36%) did not receive any treatment to the neck at any time during the course of this study.

For the same reasons already stated regarding control of the primary gingival lesion, it was impossible to ascertain accurately the efficacy of control of the metastatic lesions in the neck.

Incidence of Cervical Metastasis.—During the 10 years of this study, 55 block dissections of cervical nodes, including those done as part of a composite operation, were performed in the absence of clinically involved lymph nodes (prophylactic dissection of the neck). Histologically proved metastatic lesions were found in 11 (20%) of these 55 dissections. A total of 30 block dissections, including dissection done as part of a composite operation, were done in cases in which clinically involved lymph nodes were present; nodes were found to be actually involved in 23 instances and uninvolved in 7, an "error rate" of 23%.

Crossed Metastasis.—A total of 32 patients with lateralized primary lesions had cervical metastatic nodes examined histologically (excision of nodes or dissection of neck) at some time during this study. In 27 instances (84%), metastatic tumors appeared in the homolateral side of the neck, whereas they occurred in the contralateral aspect of the neck in 1 case (3%). In the remaining four cases (13%), metastasis was evident bilaterally; the metastatic tu-

TABLE 2.—*Squamous-Cell Carcinoma Arising in the Mandibular Gingiva: Survival Rates*

Period, Yr.	Patients		Lived Beyond Indicated Period	
	Total	Traced	Number	Per Cent *
3	118	107	67	62.6
5	94	87	45	51.7

* Based on traced patients. Inquiry as of Jan. 1, 1958. Included in the three-year group are only those patients treated three or more years prior to the time of inquiry, that is, 1954 or earlier. The five-year group includes only those patients treated in 1952 or earlier. Hospital deaths are excluded in the calculation of survival rates.

mors appeared homolaterally first in two of these cases, whereas they appeared bilaterally at about the same time in the other two. Only instances of histologically proved cervical metastasis are included in these figures, which therefore do not represent the total incidence of homolateral or contralateral metastasis, because some patients who had obvious cervical metastatic tumors were treated by irradiation without prior histologic proof of involvement.

Survival of Patients

Table 2 summarizes the survival data for these 118 patients with squamous-cell carcinoma arising in the mandibular gingiva. The five-year survival rate among the 94 patients eligible for a five-year study was 52%. The three-year survival rate for the total group was 63%.

The survival of patients according to the treatment employed was studied, taking into consideration the severity of the lesions treated. Even though the total group is fairly sizable (118 cases), the total number in each treatment group is considerably less. Thus, estimation of the merits of each method of treatment becomes difficult. When both the extent of the primary lesion and the presence of cervical metastatic tumors were considered, no significant difference in survival according to the method of treatment could be found. The small, low-grade lesions that required only treatment of the primary tumor and never involved the neck had a good prognosis. The extensive lesions that were treated by irradiation had a much less favorable prognosis, as would be expected. The large intermediate group of lesions, treated by the various methods already described, all had about an equal prognosis.

In view of the many variables encountered in this study, definite conclusions regarding the most efficacious method of treatment could not be reached. Of most significance is the five-year survival rate of 52% of the traced patients. Although this method of calculation of the survival rate is slightly dissimilar to that of other series reported, the rate does compare favorably (Table 3). Of greatest importance in comparing the rates of cure from different institutions are the factors used to govern inclusion in the study, as emphasized by Morfit in his discussion of the controversy regarding prophylactic dissection of the neck for carcinoma of the tongue; his conclusions would apply to these lesions as well as to carcinomas of the tongue. Only when large series of unselected patients treated by certain methods are used for comparison can the true worth of the procedures be evaluated. Similarly, in comparing survival data from different institutions, one must consider the years over which the treatment took place.

Comment

In the past decade, many writers have emphasized the use of extended radical operations for intraoral malignant tumors, including mandibular carcinomas. Most authors claim good success with these procedures. However, most of these extensive operations have been done relatively recently, and therefore survival data are not yet available. At the present time, only a few selected series of patients treated by these methods have been recorded in the literature. Although these radical operations probably have merit, their ultimate value in increasing the survival rate of a large series of patients remains yet to be proved.

As will be noted in Table 3, although these patients were treated by different methods, there is little difference in survival

TABLE 3.—*Cancer of the Gingiva: Reported Series*

Authors	Years of Treatment	Number of Cases	Factors of Selection	Predominant Treatment		Survival Rates		
				Primary Lesion	Neck	5-Year Determinate	5-Year Absolute	3-Year
Wilkins & Vogler	1937-1956	81 total 59 determinate 29 eligible *	17 upper 64 lower Unselected	Radiation and surgery	Surgery	9/29=31%		
Mattick & Meehan	1937-1944	118 total 101 determinate	45 upper 56 lower	Radiation	Radiation	19%		
Windeyer	1937-1946	131 total 115 treated by radiation	Floor of mouth and mandibular alveolus	Radiation	Dissection	33/115=29%		
New	Prior to 1930	187 operated on 151 traced	Lower jaw; surgical cases only	Electrocoag.	Dissection	90/151=60%	90/187=48%	
Martin	1929-1935	113	Unselected; upper and lower jaw	Surgery or radiation or both	Dissection	26%		
Lampe	1940-1951	39 total 27 eligible *	All primary lesions, all stages	Radiation	Radiation		30%	
Modlin & Johnson	1940-1950	63	All primary lesions, buccal mucosa and lower gingiva	Surgery	Surgery		22/42=52%	34/63=54%
Present study	1945-1954	118	Lower gingiva; all patients treated	Electrocoag. or surgery	Surgery	52%		63%

* Eligible = patients treated five or more years before date of inquiry.

rates among the reported series. During the period of study under consideration, the great majority of our patients were treated by excision or electrocoagulation of the primary lesion and by some type of block dissection of the neck for the cervical lymphatic structures. In many of the patients treated by electrocoagulation, block dissection of the cervical lymphatic structures and ligation of the external carotid artery preceded treatment of the primary lesion. Only a relatively few composite operations were employed during this period.

We believe that prophylactic dissection of the neck, used on an individual basis, is of value in the treatment of occult cervical metastatic tumors originating from squamous-cell carcinoma of the mandibular gingiva. In this series, the incidence of histologically involved cervical lymph nodes in clinically uninvolved necks was 20%. For lesions that are well lateralized, we believe that block dissection of the neck in the absence of clinically involved nodes is justified. The low incidence of crossed metastasis in well-lateralized lesions lends support to this principle. The problem of treatment for occult cervical metastasis admittedly becomes extremely difficult when the lesions are near the midline. For lesions in or near the midline, observation of the patients for the development of cervical metastasis is probably preferable to bilateral block dissection.

Summary

A study of 118 patients who had squamous-cell carcinoma arising in the mandibular gingiva and who were treated at the Mayo Clinic was undertaken. Definite conclusions could not be drawn regarding the most efficacious method of treatment of these lesions.

A five-year survival rate of 52% of the traced patients was obtained. It is hoped that these data, based on all cases of squamous-cell carcinoma arising in the mandibular gingiva treated at the clinic over a 10-year period, might help to establish a base line in evaluating further reported series.

Mayo Clinic.

REFERENCES

1. Erich, J. B., and Kragh, L. V.: Results of Treatment of Squamous-Cell Carcinoma of the Anterior Part of the Tongue, Am. J. Surg., to be published.

2. Erich, J. B., and Kragh, L. B.: Treatment of Squamous-Cell Carcinoma of the Floor of the Mouth, A. M. A. Arch. Surg., in this issue, page 94.

3. Lampe, I.: Radiation Therapy of Cancer of the Buccal Mucosa and Lower Gingiva, Am. J. Roentgenol. 73:628-638 (April) 1955.

4. Martin, H. E.: Cancer of the Gums (Gingivae), Am. J. Surg. 54:765-806 (Dec.) 1941.

5. Mattick, W. L., and Meehan, D. J.: Carcinoma of the Gum, Surgery 29:249-254 (Feb.) 1951.

6. Modlin, J., and Johnson, R. E.: The Surgical Treatment of Cancer of the Buccal Mucosa and Lower Gingiva, Am. J. Roentgenol. 73:620-627 (April) 1955.

7. Morfit, H. M.: Neck Dissection in Treatment of Cancer of the Tongue and Mouth, in Current Surgical Management: A Book of Alternative Viewpoints on Controversial Surgical Problems, edited by J. H. Mulholland, E. H. Ellison, and S. R. Friesen, Philadelphia, W. B. Saunders Company, 1957, pp. 387-392.

8. New, G. B.: Malignant Disease of the Mouth and Accessory Structures, Am. J. Surg. 30:46-52 (Oct.) 1935.

9. Wilkins, S. A., Jr., and Vogler, W. R.: Cancer of the Gingiva, Surg. Gynec. & Obst. 105: 145-152 (Aug.) 1957.

10. Windeyer, B. W.: Carcinoma of the Floor of the Mouth, Brit. J. Surg. 41:231-237 (Nov.) 1953.

Severe Gastrointestinal Hemorrhage Resulting from Recklinghausen's Disease

TURLEY FARRAR, M.D., and FENWICK W. CHAPPELL, M.D., Memphis, Tenn.

Although intestinal neurofibromatosis associated with Recklinghausen's disease is a rare condition, it may cause severe gastrointestinal disturbances as a result of hemorrhage or perforation. Shaiken [1] and Shaw [2] have each reported a single case of exsanguinating gastrointestinal hemorrhage resulting from neurofibromata of the small intestine in Recklinghausen's disease. Warshauer and Nelson [3] reported a case in which a neurofibroma in the jejunum perforated. Hartman [4] reported a case of fibromyxosarcoma of the stomach in a patient with Recklinghausen's disease which he believed arose from a neurofibroma of the stomach wall. River et al.,[5] in a collective review of the world literature on benign tumors of the small intestine, including 1,399 cases, found only 90 cases of neurogenic tumors of all types. In only 14 of these cases were there cutaneous neurofibromata (Recklinghausen's disease).

Report of a Case

A 50-year-old white man was admitted to the surgical service of Baptist Memorial Hospital on Dec. 28, 1955, in a state of shock because of vomiting blood and passing tarry stools. Twenty-four hours prior to admission he had passed a large, dark stool, and eight hours before admission he started vomiting bright blood and again passed copious dark, bloody stools. His local physician had given him a vasopressor drug and started him on intravenous infusion of dextrose before getting him on his way to the hospital. He had had no previous symptoms referable to the gastrointestinal tract except for occasional mild epigastric pain after meals. There was no history of hematemesis or melena before this illness. He further stated that he had had several nodular tumors of the skin for many years and had been told they were benign. He had been discharged from the orthopedic service of this hospital about a month previously, at which time he was treated for traumatic compression fractures of vertebrae L1 and L2. Recovery from this injury had been uneventful except for a mild degree of abdominal distention and ileus during the first few days of the illness.

The family history was noncontributory. No other members of the family were known to have skin tumors such as his.

On admission the patient appeared pale and apprehensive and complained of moderate epigastric pain. His pulse rate was 144 a minute; the blood pressure was 118/80. Inspection and examination of the skin revealed numerous soft, nodular skin tumors and café au lait spots. There were a hairy mane on the back and an elephantiasic neuroma involving the skin and subcutaneous tissue of the left foot and ankle (Fig. 1). The abdomen was slightly distended; there were no palpable abdominal masses or tenderness. Peristalsis was nor-

Fig. 1.—Photograph made postoperatively.

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From the Department of Surgery, Baptist Memorial Hospital.

mal. Rectal examination was negative except that old blood was noted on the examining finger. The hematocrit was 26 vol. %.

The presumptive diagnosis was upper gastrointestinal hemorrhage, probably due to duodenal ulcer. He was started on supportive therapy, consisting of a soft bland diet, sedation, intravenous fluids, and blood transfusions. After two days he seemed to be responding satisfactorily to treatment, and there was apparent cessation of bleeding. The hematocrit was 36 vol. %. On the third day the patient again vomited blood, passed bright blood from the rectum, and went into shock. After a favorable response to transfusions, an upper gastrointestinal roentgenogram was made, using a thin barium meal. The esophagus appeared to be normal, but the stomach and duodenum were filled with mottled, particulate matter, presumed to be blood clots. No filling defect either in the stomach or in the duodenum could be demonstrated by roentgenogram. During the next 24 hours he continued to vomit blood and pass tarry stools, and in spite of multiple transfusions he remained in a state of mild shock.

On the fourth day after admission an emergency laparotomy was performed in an attempt to find and stop the source of bleeding. On exploration, multiple smooth round nodular masses were observed throughout the duodenum and upper jejunum. These mases ranged from 0.3 to 2.5 cm. in diameter. No tumors were found in the stomach, distal jejunum, ileum, or colon. A gastroduodenotomy revealed no active bleeding in the stomach and no definite bleeding points in the duodenum, although the duodenum was filled with fresh blood. Although no ulcerated area was noted in the mucosa overlying the tumors, it was felt that the bleeding was coming from the region of the first portion of the duodenum, and two large nodules were locally excised at that point. Several tumors in the upper jejunum were also excised. Firm nodular tumors which were palpated behind the duodenum at that time were interpreted as being retroperitoneal neurofibromata, and probably not significant.

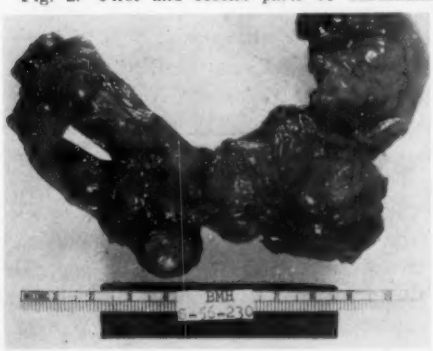
The pathologic on these tumors which were removed was that of neurilemmomas, with no evidence of malignant change.

In the immedate period following surgical exploration, the patient showed improvement for two days, although multiple transfusions were necessary to maintain the hematocrit above 35 vol. %. On the third day after operation a massive hemorrhage again occurred. The patient vomited bright blood and passed tarry stools, and again he went into shock. During the next few days the bleeding continued, and even though repeated transfusions were given, the hematocrit continued to drop, reaching 21 vol. %. On the eighth postoperative day the patient was again operated upon. At this time the entire duodenum was mobilized, and several tumors were found in the wall of the first and second portions of the duodenum; the largest of these was approximately 5 cm. in diameter and was located just above the ampulla of Vater. The mucosa overlying this tumor was superficially ulcerated (Fig. 2). No active bleeding was seen at this time. With the common bile duct opened and a small probe threaded through the ampulla, the first part and a portion of the second part of the duodenum were excised, along with the ascending duodenum and approximately 3 ft. (90 cm.) of the supper jejunum. The proximal duodenal stump was closed, special care being taken not to damage the common duct. The jejunum was brought up beneath the ligament of Treitz and anastomosed to the distal end of the duodenum. Following this, the antrum of the stomach was then resected, and an antecolic gastrojejunostomy was performed after the method of Hofmeister (Fig. 3). A long-limbed T-tube was left in the common duct.

The pathologic diagnosis was neurofibromata of the duodenum and jejunum.

The patient's course after this operative procedure was satisfactory. There was slight excoriation around the T-tube, where apparently there was some leakage of bile and pancreatic secretions. The T-tube was removed on the 16th day, and

Fig. 2.—First and second parts of duodenum.



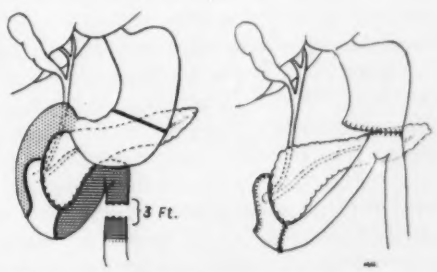


Fig. 3.—Operative procedure. Shaded areas represent involved bowel.

drainage stopped within 48 hours after its removal. He was discharged a few days later. He had been followed in the outpatient department at regular intervals; and he stated on his last visit, on Jan. 15, 1958, that he had had no recurrence of bleeding episodes. He has gained some weight, and his progress has been very satisfactory.

Conclusions

Patients with cutaneous neurofibromata who have vague gastrointestinal complaints should be carefully examined for gastrointestinal tumors. Since roentgen examination of the small bowel is seldom helpful in establishing a diagnosis, the persistence of symptoms is indication for early surgical exploration.

Suite 306, Baptist Medical Building, 20 South Dudley St. (3).

REFERENCES

1. Shaiken, J.: Tumors of the Small Bowel, Am. Pract. & Digest Treat. 3:388-392, 1952.

2. Shaw, R. C.: von Recklinghausen's Disease of the Small Intestine Associated with Skin Lesions, Am. J. Surg. 80:360-363, 1950.

3. Warshauer, F. B., and Nelson, R. E.: Neurofibroma of Jejunum with Perforation: A Case Report, Am. Surgeon 19:467-472, 1953.

4. Hartman, H.: Fibromyxosarcoma of the Stomach in a Case of Neurofibromatosis (von Recklinghausen's Disease), Surg. Gynec. & Obst. 44:308-310, 1927.

5. River, L.; Silverstein, J., and Tope, J. W.: Benign Neoplasms of the Small Intestine: Critical Comprehensive Review with Report of 20 Cases, Collective Review, Internat. Abstr. Surg. 102:1-38, 1956, in Surg. Gynec. & Obst., Jan., 1956.

Sliding Inguinal Hernia in Female Infants

STEPHEN L. GANS, M.D., Beverly Hills, Calif.

In recent years, the literature on the subject of infant hernias has been voluminous. One phase of this problem, sliding hernia in female infants, has received little attention. In the older literature isolated mention of this sliding hernia is made without adequate descriptions or methods of treatment. In more recent reviews of the subject of infant hernias, this particular type of infant hernia is completely ignored.[1,3,5,7,10] In 1953 Roberto[11] described a sliding hernia, with uterus, adnexa, and bladder making up one wall of the sac, in a 5-week-old infant. In 1957 Wiley and Chavez[13] reported 12 cases of sliding hernia out of a total of 52 female infant hernias and described their method of correcting this condition. Koop,[6] in 1957, and DeBoer,[2] in 1957, in their general reviews of infant hernias, state that many of the female infants had sliding hernias, and DeBoer describes his method of treatment. Goldstein and Potts[4] and Swenson[12] have finally put this subject in the proper perspective. Our paper supports and amplifies their observations.

The source of the material for this report is a series of 400 infants and children, having 516 inguinal hernias. The difference between these two figures represents the number of patients with bilateral hernias. Of the total, there were 68 female patients (17%) with 90 hernias. The 19 sliding inguinal hernias in female infants represents 28% of all hernias in female infants in the series. From these figures it becomes apparent that this condition is a relatively common one which merits adequate consideration, particularly because it has not been sufficiently recognized in the past.

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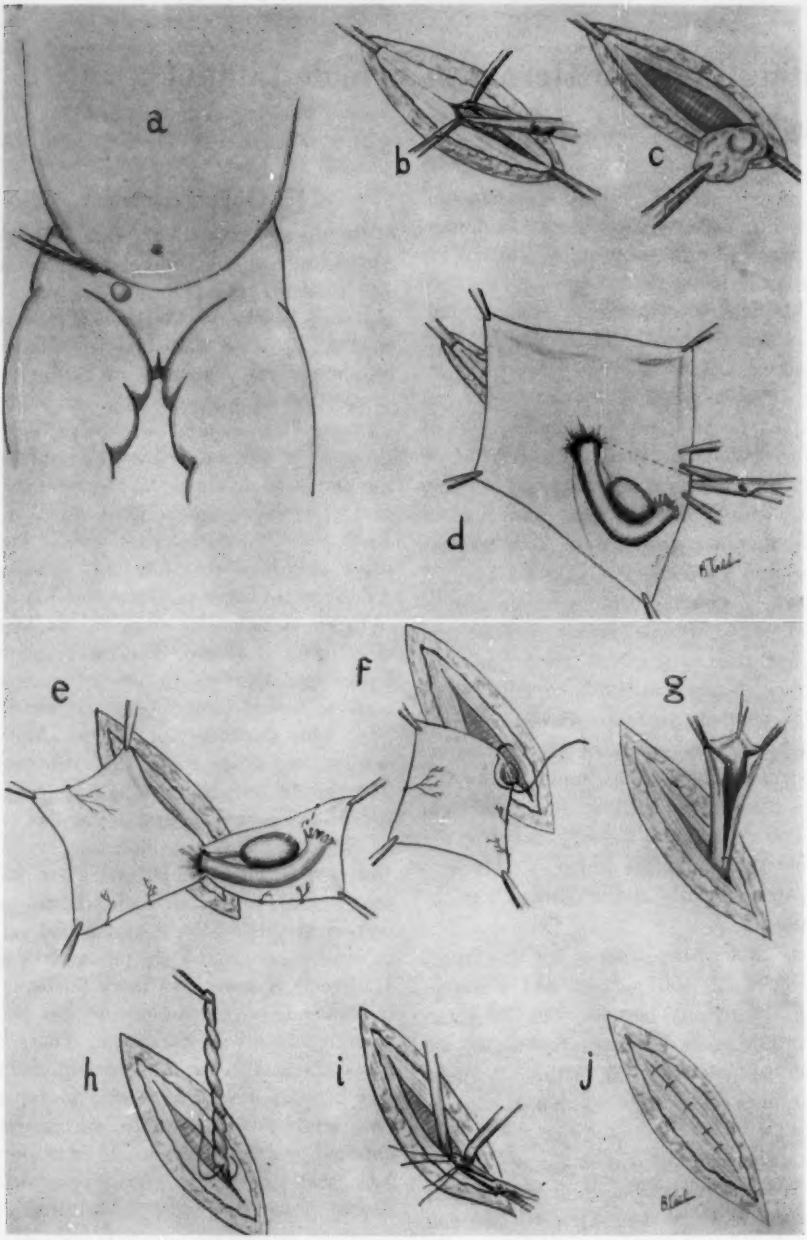
Clinical Summary

Of the 19 infants, 17 were less than 4 months of age. One was 9 months old, and one returned at the age of 3½ years with a sliding hernia on the left side, after having had an incarcerated sliding hernia repaired on the right side at the age of 6 weeks. In the majority of cases there was no complaint. A mass was palpated in the groin or labium majus by the mother or by the pediatrician during a routine examination. The asymptomatic mass varied in size from 1 to 2.5 cm. in diameter. It was always somewhat movable, and occasionally reducible. In other instances the infant was irritable, occasionally drew up its legs, as if in pain, and the mass was somewhat tender to palpation. In two instances there was the typical picture of severe incarceration, with swelling, tenderness, pain, distention, vomiting, fever, and dehydration. The differential diagnosis is from inguinal lymphadenopathy, hydrocele of the canal of Nuck, and male pseudohermaphroditism. Inflamed inguinal lymph nodes are usually multiple and irregular, and a focus of infection is frequently demonstrated on the extremity or around the anus or vagina. Hydrocele is sometimes more fluctuant and transmits light, but frequently can be differentiated only at operation. There were two such cases in our entire series, and each was associated with a hernia, as expected. One infant with apparently normal female external genitalia had testicles in the hernial sacs, and laparotomy revealed no demonstrable internal genital structures. This condition represents a case of male pseudohermaphroditism.

At surgery it was demonstrated in every case of sliding hernia that the Fallopian tube and the adjoining part of the broad ligament made up at least a portion of the



(*a*) Incision in lateral portion of the suprapubic crease. (*b*) Incision of the external oblique aponeurosis. (*c*) Demonstration and development of the hernia sac. (*d*) The sac is opened and the sliding element is outlined. (*e*) The sac is incised on each side of the sliding element, and hemostasis is secured in the edges. (*f*) The sliding element is tucked in through the internal ring, and the neck of the sac is reconstituted with a silk suture (*g*). (*h*) The sac is twisted and suture-ligated at its neck. (*i*) After amputation of the sac, the internal ring is closed by placing sutures in the transversalis fascia. (*j*) The external oblique aponeurosis is closed.

hernia-sac wall. Usually the ovary was also included, and occasionally a portion of the uterus or the entire supracervical part of the uterus. This series does not include those instances in which these structures were drawn up into the sac by vigorous traction. In most instances the organs involved were normal. In two patients the involved ovary was discolored and edematous but was returned to the peritoneal cavity. In one patient a segment of ileum was also discolored, but was demonstrated to be viable and was preserved.

Surgical Treatment

The principle involved in surgical treatment is the same as that involved in treating all infant hernias—complete removal and high ligation of the hernia sac. It is our contention that this cannot be done without special handling of the sliding portion of the sac, as will be described.

A 3 cm. transverse incision is made through the skin and subcutaneous tissues of the lateral portion of the suprapubic crease. The external oblique aponeurosis is incised in the direction of its fibers and retracted, carefully preserving the ilioinguinal nerve. The hernia sac is demonstrated, dissected out, and carefully opened in an area away from the sliding elements. The sac is then incised on each side of the sliding element down to the neck. After securing hemostasis in the edges, the tongue of sac, with the sliding element, is tucked through the internal ring into the peritoneal cavity. A silk suture is then placed through the cut edges of the sac, making its circumference complete again. The sac can now be handled in the usual fashion by high ligation and amputation, without injuring adnexal structures. We accomplish this by twisting the empty sac and suture-ligating it at its neck with silk. Another fine silk suture or two is used to approximate the transversalis fascia at the internal ring. The external oblique aponeurosis is closed with fine interrupted silk or surgical gut. The subcutaneous tissues and Scarpa's fascia are approximated with 00000 plain surgical gut, and the skin edges are neatly approximated with a continuous subcuticular 00000 plain surgical gut suture. A small dressing is applied. The infant is discharged on the same day or on the following morning, depending on the completeness of recovery from anesthesia. No particular restrictions are placed on the patient's activity.

Embryology

Study of the embryological background makes apparent the reason for this anomaly. The urogenital system as a whole arises in the dorsal body wall. When the mesonephros begins to grow rapidly in bulk, it bulges out into the celom, pushing ahead of itself a covering of peritoneum. At both ends of the mesonephros the peritoneum is, in this process, thrown into folds. The cephalad fold extends to the diaphragm and is known as the diaphragmatic ligament of the mesonephros. The distal fold extends to the caudal end of the celom and is called the inguinal ligament of the mesonephros. The gonads develop and the mesonephroi degenerate until there remains a pair of mesenteries suspending the Müllerian ducts in their ventral margins and the ovaries on their mesial faces. Farther caudally the Müllerian ducts fuse with each other in the midline to form the uterus, and the supporting peritoneal folds coalesce medially to form part of the broad ligament supporting the uterine tubes. As the gonads increase in size, they sag farther into the peritoneal cavity, and the ovaries move laterally. In this process the inguinal ligament of the mesonephros becomes angulated, the cephalic part becoming the round ligament of the ovary, and the caudal part becoming the round ligament of the uterus. The caudal end of the round ligament is embedded in the connective tissue of the labium majus in a position analogous to the anchorage of the gubernaculum of the male. In the meantime, a celomic evagination, known as the canal of Nuck, is formed

in the region of each side where the round ligament is attached. In our opinion this is the place and time that the sliding hernia is formed. If the evagination occurs at the site of the broad ligament or its primordium, it will be unable to close because of the presence in it of adnexal structures. Another possible explanation of the occurrence of the sliding hernia is that the round ligament of the uterus becomes foreshortened and draws with it the broad-ligament structures into the inguinal canal and labium. This is analogous to the explanation by some of the normal descent of the testis into the scrotum. Whichever theory is adhered to, the end-result is an open processus, one aspect or wall of which is made up by the broad ligament or a reflection of it; therefore it contains within its lumen the uterine tube, frequently the ovary, and occasionally the uterus.

Comment

In general, the same principles apply to this type of hernia as apply to all infant hernias. Surgical correction should be carried out without delay. In addition to the usual dangers of incarceration and strangulation are the dangers of ovarian atrophy or tubal stricture. Any operation for correction of infant hernia should be primarily concerned with complete removal of the hernia sac. There are four different methods of carrying out this principle in this type of sliding hernia. The first involves dissecting the broad-ligament structures away from the peritoneum so that the whole sac may be removed. This is extremely difficult to do without considerable bleeding and possible trauma to the delicate organs. The second method invokes the principle described by LaRoque,[8] that of making a separate opening into the peritoneal cavity and drawing the sac into the peritoneal cavity from the inside. We feel that this method is too complicated and involves more surgery than is necessary. A third method handles the problem by tucking the whole sac and its contents into the peritoneal cavity and then placing concentric purse-string sutures at the neck. This method may have merit, but we have always experienced great difficulty in placing purse-string sutures in the neck of any filmy hernia sac, even under direct vision; and we feel that placing them blindly after tucking in the sac may not produce the desired effect, and may even be hazardous to the blood supply of the gonadal structures. The fourth method is the one described above in detail. It appears to us to be a simple, direct method for completely removing the hernia sac, while carefully preserving the gonadal structures.

Summary

Sliding inguinal hernia in the female infant is a relatively common condition, which has not been adequately recognized in the past. In our series, 28% of all female infants and children with inguinal hernias had sliding hernias. This approximates the only other published statistics.

The diagnosis is usually made by palpating a mass during routine examination. Occasionally the complete picture of an incarcerated hernia prevails.

Treatment consists of complete removal and high ligation of the sac. A satisfactory method for accomplishing this is described in detail.

9735 Wilshire Blvd.

REFERENCES

1. Clatworthy, H. W., Jr., and Thompson, A. G.: Incarcerated and Strangulated Inguinal Hernia in Infants: A Preventable Risk, J. A. M. A. 154:123-126 (Jan. 9) 1954.

2. DeBoer, A.: Inguinal Hernia in Infants and Children, A. M. A. Arch. Surg. 75:920-927 (Dec.) 1957.

3. Duckett, J. W.: Treatment of Congenital Inguinal Hernia, Ann. Surg. 135:879-885 (June) 1952.

4. Goldstein, I. R., and Potts, W. J.: Inguinal Hernia in Female Infants and Children, Ann. Surg. 148:819-822 (Nov.) 1958.

5. Gross, R. E.: The Surgery of Infancy and Childhood, Philadelphia and London, W. B. Saunders Company, 1953.

6. Koop, C. E.: Inguinal Herniorrhaphy in Infants and Children, S. Clin. North America 37:1675-1682 (Dec.) 1957.

7. Kristiansen, C. T., and Snyder, W. H., Jr.: Inguinal Hernia in Female Infants and Children, West. J. Surg. 64:481-484 (Sept.) 1956.

8. LaRoque, G. P.: The Intra-Abdominal Method of Removing Inguinal and Femoral Hernia, Arch. Surg. 24:189-203 (Feb.) 1932.

9. Patten, B. M.: Human Embryology, Ed. 2, New York, Blakiston Company (Division of Doubleday & Company), 1953.

10. Potts, W. J.; Riker, W. L., and Lewis, J. E.: Treatment of Inguinal Hernia in Infants and Children, Ann. Surg. 132:566-576 (Sept.) 1950.

11. Roberto, A. E.: Unusual Inguinal Hernia in Infancy, New York J. Med. 53:3044 (Dec. 15) 1953.

12. Swenson, O.: Pediatric Surgery, New York, Appleton-Century-Crofts, Inc., 1958.

13. Wiley, J., and Chavez, H. A.: Uterine Adnexa in Inguinal Hernia in Infant Females: Report of a Case Involving Uterus, Both Uterine Tubes and Ovaries, West. J. Surg. 65:283-285 (Sept.-Oct.) 1957.

Primary Carcinoma of the Liver

Case Report and Review of Literature

CAPT. DAVID P. MINICHAN Jr. (MC), U. S. A. F.; ROBERT L. A. KEELEY, M.D.; HUGH H. TROUT Jr., M.D., and DANIEL LEAVITT, M.D., Roanoke, Va.

Primary carcinoma of the liver is comparatively rare in this country. Few cancers, however, have a poorer prognosis once the disease is recognized. The disease apparently spreads silently through the blood-filled sinusoids, destroying much parenchyma before it causes sufficient damage to make its presence known. Hope of cure depends on early diagnosis and complete surgical extirpation of the tumor. Recent advances in surgical technique have brightened the possibility of cure. If these advances are to be fully utilized, however, some method of earlier diagnosis must be discovered.

Presented here is a case of malignant cholangioma in a young woman which was discovered in her second postpartum month. A total right hepatic lobectomy was carried out in an attempt to eradicate the tumor.

One cannot say who first described primary liver cancer. Carl von Rokitansky,[45] in 1849, insisted that primary liver cancer was a common affection. He also separated primary from secondary tumors of the liver, apparently on the basis of grossly excluding other organ sites rather than by histologic examination.[15]

Virchow warned that before a diagnosis of primary liver cancer could be made a careful search of the eye, breast, and gastrointestinal tract should be made, since he believed that an organ which is a frequent site of metastatic disease is rarely the site of a primary cancer. This, of course, is true for the liver but incorrect for the lung and brain.[15] In 1879, Kelsch and Kiener[34] reviewed the literature and found only one case of primary liver cancer which they considered authentic. To this they added two cases of their own. In 1881, Sabourin[49] added four more cases. Prior to the time of Sabourin, primary liver cancer was considered commoner than secondary cancers; hence, statistics gained from many early case reports and death certificates are invalid because there was no histologic confirmation.[10]

By 1901, Eggel[16] had found 163 cases, and in 1939, Charache's[9] exhaustive search of the world literature produced 1,125 cases. Since then, many excellent papers have contributed additional information to the study of this disease.

Incidence

Primary carcinoma of the liver holds a unique position among human cancers in its geographical and racial distribution. All evidence indicates that the incidence is highest in countries inhabited by yellow-skinned and dark-skinned races and lowest in the United States and the Scandinavian countries. Data collected from some of the larger clinics in this country show the autopsy incidence of this disease varies from 0.02% to 0.44%, with an average of 0.26%.* The general incidence in Europe is about the same as in this country, namely, 0.20%, but is considerably higher in Greece (1.0%) and Italy (0.50%).[45]

The highest reported incidence is in the Bantu of Portuguese East Africa, where

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Department of Surgery, Jefferson Hospital.

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* References 4, 13, 19, 26, 32, 36-39, 42, 46, 47, 51, 60.

primary carcinoma of the liver accounts for 50.9% of all malignancies. In the semi-Bantu tribes who inhabit the west coast of Africa, this disease represents 18.7% of all cancers. Berman [4] reported that among the Bantu workers at the Witwatersrand gold mines primary liver carcinoma accounted for 90.5% of all cancers. These workers were all males and ranged in age from 16 to 40 years.

Tull [62] found 134 cases of primary liver cancer in 17,664 autopsies at the Tan Tock Sing Hospital in southern China, for an autopsy incidence of 0.92%, or 33% of all types of cancer. This frequency is about the same for the Orient in general.[29,33,64]

From Vancouver, B. C., Canada, Strong and Pitts [58] reported nine cases of primary liver carcinoma, eight of whom were Chinese who had emigrated from the Kwangtung Province of southern China.

Wilbur, Wood, and Willett [68] found 19 Chinese with primary liver cancer out of 49 cases in San Francisco. This seems very significant in view of the fact our population has relatively few Chinese. Most of these patients were apparently born in China. Whether the high incidence will persist in future generations of American Chinese remains a question for the future.

These observations illustrate the principle of the transportation of carcinogenic influences either in the form of already biased liver cells or of environmental carcinogenic factors.[15]

The composite autopsy incidence for primary carcinoma of the liver in American Negroes of 0.44% is somewhat higher than the incidence of 0.26% calculated for the general population.[45] Berman [5] suggests that it is possible that Negroes carry with them a predisposition to the disease when they leave their native habitat. Kennaway,[35] Lynch,[38] and Steiner [56] believe the available data supply no evidence that the American Negro is especially susceptible to the disease.

Throughout the world, primary liver cancer is predominantly a disease of males. In areas where the incidence is low, males slightly outnumber females, but in areas where the incidence is high, males greatly outnumber females.[15] In almost two-thirds of Eggel's [16] cases, the patients were males, and Hoyne and Kernohan [32] reported that 74.2% of their cases were males. In Greene's [24] series, males outnumbered females six to one.

Liver carcinoma is primarily a disease of the older age group, though it may occur at any age. It is most frequently seen between the ages of 40 and 60 and especially between 50 and 60.[17,32,46,55] Bigelow and Wright [6] found 95 childhood cases from the literature, 55 of which appeared before 2 years of age and 35 before 1 year. Noeggerath [43] observed a case of primary liver carcinoma in a newborn infant, and Widerhofer [67] reported a case in a 3-day-old child.

In areas where the incidence is high, the greatest incidence is in younger age groups. Thus, in Berman's study of the Bantu, 82.6% of his cases were under 40 years of age.

Pathology

Before a diagnosis of primary liver carcinoma can be established at laparotomy, a careful search must be made for a primary focus. At times, hepatic metastasis from primary lesions elsewhere may far overshadow the original tumor, and should the latter be missed, an erroneous diagnosis of primary liver cancer might be made. Particular attention should be directed to the extrahepatic bile ducts, gallbladder, kidneys, adrenals, breasts, and gastrointestinal tract.

Primary liver carcinoma may be found in one of three forms as regards the gross appearance: (1) massive, (2) nodular, or (3) diffuse.

(1) *Massive.*—In this form, the liver is usually enlarged by a single large, irregular tumor mass involving one lobe, usually the right. Surrounding the large mass may be several smaller satellite tumor nodules. The

size, color, and consistency of the tumor mass may vary greatly. The tumor may be fluctuant and filled with liquid necrotic material, which may suggest a liver abscess. When necrosis occurs near Glisson's capsule, adhesions may exist between the tumor and adjacent viscera.[45]

(2) *Nodular.*—Here the liver is studied with numerous tumor nodules of different sizes, many of which project through Glisson's capsule. The nodules may be firm, soft, or even cystic as the result of necrosis. The color may vary from a translucent gray-white to a yellow, pink, red, purple, or even dark brown. The color depends upon the degree of maturity of the liver cells, the presence of bile, degeneration, necrosis, and/or hemorrhage. In both the massive and nodular forms, the tumor grows in an expansile fashion, crowding and compressing the adjacent liver parenchyma.[15] In both forms, the liver is usually greatly enlarged.

(3) *Diffuse.*—This is also frequently called infiltrating or carcinomatous cirrhosis. The liver is extensively scarred with many tiny nodules and may be normal in size or shrunken. The color varies from a gray-white to yellow, tan, or light brown. The liver may so closely resemble a typical Laennec's cirrhosis that it is impossible to differentiate the two grossly.[2]

It is not always possible to differentiate grossly between primary liver carcinoma and metastatic disease, and the final diagnosis rests with the pathologist. In contrast to metastatic cancer, the liver nodules of primary carcinoma are only rarely umbilicated.

Primary carcinoma of the liver may be divided into three major histologic groups: (1) liver-cell type (malignant hepatoma), (2) bile duct-cell type (malignant cholangioma), and (3) atypical, or mixed, type (malignant cholangiohepatoma).

Liver-Cell Carcinoma.—This is the commonest form of liver cancer. The tumor arises from the polygonal cells; hence, the tumor cells are polygonal or polyhedral in shape. The nuclei show much variation in size, shape, and staining reaction. Multiple nucleoli and bizarre mitosis may be seen. There is abundant granular cytoplasm which usually has acidophilic staining characteristics. The cells often have the ability to secrete bile, and intracellular bile pigment is frequently seen in the cytoplasm. The cells are arranged in sheets or cords, and multinucleated giant cells may be found. A cirrhotic reaction is common. The tumor cells are separated by a capillary stroma, which is important in differentiating liver-cell from bile duct-cell carcinoma.

Bile Duct-Cell Carcinoma.—This type arises from the epithelium of the intrahepatic bile ducts. The cells are cuboid or cylindric, with small, hyperchromic nuclei. The cytoplasm is clear but frequently has a basophilic staining reaction. The cell morphology is more constant, as compared with liver-cell carcinoma, with giant cells, mitosis, and bizarre cell types being rarely seen. Intracellular bile pigment is not found, but inspissated bile may be seen in the bile duct lumens. The tumor cells tend to form glands or ducts and are supported by a fibrous, connective tissue stroma which has relatively few capillaries. The fibrous tissue tends to become denser, compressing the tumor nodules, giving the appearance of being encapsulated. While necrosis is frequently seen in nodules of liver-cell carcinoma, this is not a prominent feature of bile duct-cell carcinoma.

Atypical, or Mixed, Type Liver Carcinoma.—This form has tumor cells characteristic of both the malignant hepatoma and the malignant cholangioma. There is usually a predominance of the bile duct cells, since these tend to grow and spread more rapidly.[60]

Liver-cell carcinoma is predominantly a disease of males. In Eggel's[16] series, 57% of his patients with bile duct-cell carcinoma occurred in women. Hoyne and Kernohan[32] reported 11 cases of malignant cholangioma, 6 of which were in women. Cunningham,[13] however, found only two or six cholangio-

mas in women. Fried [19] believes this is due to the frequency of cirrhosis in males and biliary tract disease in females.

While intrahepatic spread is almost the rule, extrahepatic metastases are present in roughly half the cases that come to autopsy.[5,9,24,31,68] In the series reported by Rosenberg and Ochsner,[46] however, spread beyond the liver was found in 40 of 55 cases. Ewing [17] mentioned that extrahepatic metastases are seen earlier and more frequently in malignant hepatoma than in malignant cholangioma. This would seem logical, since liver-cell carcinomas tend to invade blood vessels earlier than bile duct-cell carcinomas. This observation is to be seriously questioned, and, if accepted at all, it must be with reservation.[2,32]

Metastasis takes place through the portal and hepatic venous channels and, to less extent, through the lymphatics. The most frequent sites of extrahepatic spread are to the regional lymph nodes (hilar, periportal, mesenteric, retroperitoneal, periaortic, mediastinal, and gastrohepatic) and the lungs. The lungs are especially vulnerable, since the tumor cells may reach them not only via the blood stream or lymphatics but also by direct extension through the diaphragm. Although no organ is immune to metastatic attack, some of the commonest sites of metastasis are the portal venous system; bones, especially the ribs and vertebrae; spleen; brain; kidneys; adrenals, and pancreas. The disease may spread to the stomach, gallbladder, duodenum, colon, or omentum by direct extension. Contrary to what one might expect, generalized peritoneal seeding is infrequently seen.

Tumorous thrombosis of the large veins is frequently found, since malignant liver cells early invade the vein walls. Eggel [16] found tumorous thrombosis in the large veins in 20% of his series, and in 1939, Gregory [25] studied 12 cases of primary liver carcinoma with tumorous thrombosis of the inferior vena cava. Gelfand [21] believed that the serosanguinous ascitic fluid found in primary liver cancer was probably due to portal vein thrombosis. It has been demonstrated, however, that the ascitic fluid is not necessarily bloody when tumorous thrombosis of the portal vein is present.[46] Rapid ascites, the appearance of hemorrhoids, and dilated veins over the abdomen lead one to suspect this complication.

In primary liver carcinoma, tumorous thrombosis of the inferior vena cava is more frequently seen than portal vein thrombosis. The large thrombus, which usually contains clumps of malignant cells, may propagate proximally into the right atrium. When this occurs, there is noted an increase in dyspnea, engorgement of the jugular veins, increased venous pressure in the arms, and a rapid accumulation of ascitic fluid.

The metastatic nodules of both liver-cell carcinomas and bile duct-cell carcinomas tend to display the same architecture as the parent tumor. Metastatic lesions of malignant hepatomas may show bile secretion.

The vast majority of liver carcinomas are of unicentric origin, although liver cancers of multicentric origin have undoubtedly occurred. Advocates of the multicentric theory reason that a previously damaged liver has numerous sites of potential carcinogenesis, and they point out that frequently in livers with widespread metastatic nodules no direct continuity can be demonstrated between the nodules on serial sections. Most investigators believe in the unicentric theory of origin, since the original cancer invades the portal system very early, scattering metastases throughout the liver.

Etiology

In discussing the possible etiology of primary liver cancer, one should consider the distribution of this disease in nature. Not only is it found in all races of man, but it is found in many species of animals as well. Liver cancer has been reported in domestic beasts, cats, dogs, fowls, wild ducks [8] and even a white-tailed deer.[69]

Cirrhosis.—Cirrhosis is both a precursor and a consequence of primary liver carci-

noma, but the exact relation is not known. In roughly two-thirds of the reported cases of primary liver carcinoma there is associated cirrhosis. In many series, the percentage is much higher. In fact, Counseller and McIndoe[10] question the authenticity of a diagnosis of primary carcinoma of the liver in the absence of cirrhosis. The reparative processes which usually follow damage to the liver may tend to overgrowth or actual hyperplasia. These compensatory hyperplastic nodules may eventually undergo malignant changes. The reason that one hyperplastic nodule may remain benign while another becomes malignant is not known.[32]

Berk and Lieber[3] studied 1,989 cases of cirrhosis, 90 (4.5%) of which were associated with primary liver cancer. In each case, there was strong evidence that the cirrhosis preceded the cancer. Cirrhosis is more frequently associated with liver-cell carcinoma than bile duct-cell carcinoma.

In children, primary liver cancer and cirrhosis have a less definite relationship. In Steiner's[55] 77 cases of primary carcinoma of the liver in children, only 2 had a concomitant cirrhosis and both were in older children.

Although the portal cirrhosis of Laennec is most commonly associated with carcinoma of the liver, any type of cirrhosis may be found. Among those reported with liver cancer are posthepatitis cirrhosis, post-toxic cirrhosis, cholangitic biliary cirrhosis, syphilitic cirrhosis, and various parasitic cirrhoses.

Dietary Factors.—The most recent advances in the study of liver cancer have been in the field of nutrition. In geographic areas where the incidence of primary liver carcinoma is high, there is an associated high incidence of cirrhosis as well as deficient nutrition.

Gilbert and Gillman[22] found cirrhosis of the liver in four out of five autopsies performed on male Bantu at the Johannesburg General Hospital. The Bantu are extremely poor and live on the cheapest available carbohydrate, namely, corn. Meat is a luxury and is eaten only on ceremonial occasions or when their sheep or cattle die of disease. Fermented milk also forms a large part of their diet. The average Bantu diet is, therefore, high in fat but low in protein and vitamins. Gilbert and Gillman[22] showed that rats fed a diet similar to that of the Bantu developed frank cirrhosis in 20%.

The evidence now indicates that carcinoma of the liver in these people is not the result of a deficient diet, nor do such diets induce cancer, but that in long-maintained dietary deficiencies, certain factors are lacking which act as defense mechanisms against malignancy.[48]

Chemicals.—Japanese investigators[72] found that primary liver cancer could be induced in rats and mice by the addition of certain azo dyes, including dimethylamino-azo-benzene, or butter yellow, the artificial coloring used in foodstuffs. These experiments were repeated in Europe and the United States, and the results failed to show the high incidence of liver carcinoma. Further investigation showed the discrepancy was accounted for by the fact that the Japanese animals were fed a stock diet of rice, while the animals in the American and European laboratories were fed a stock diet built around wheaten cereals.[11]

Opie[44] showed that fat in the diet accelerated the production of liver tumors by butter yellow. Also, rice favors the production of primary hepatic tumors by agencies that are not yet explained. Butter yellow produces hepatic tumors in the absence of cirrhosis, but with diets that produce cirrhosis, the frequency of tumors increases with the severity of the cirrhosis.

It has now been demonstrated that in rats who were fed butter yellow, if the amino acids, cystine and choline, and the B-complex vitamins were added to the diet, the incidence of liver carcinoma was considerably lower. It would seem, then, that these factors afford some protection to the liver against the formation of tumors.[28,40]

Cramer [11] states that in the etiology of liver cancer there are two remote causes: a carcinogenic agent, butter yellow, and the susceptibility of the liver to that agent. In this case, the susceptibility factor is not inherited or transmitted from the parent but is dependent on conditions acting on the organism from without, namely, the diet. Thus far, no identification has been made of the carcinogenic agent that is responsible for the prevalence of primary liver carcinoma among the natives of the Orient, but the dietary factor predisposing the liver—and only the liver—to the development of cancer is the same for rats and for human beings. Cramer adds that it is justifiable to conclude that liver cancer, as it occurs among the natives of the Far East, can be prevented by dietary measures.

Parasites.—Practically every parasite which involves the liver has been reported in association with primary liver carcinoma. This is particularly true of the blood fluke, Schistosoma, and the liver flukes, Clonorchis sinensis and Opisthorchis felineus.

Strong, Pitts, and McPhee,[59] in a series of autopsies performed on Chinese at the Vancouver General Hospital, reported a significant association of livers infested with C. sinensis and primary liver cancer. Each of their Chinese patients had emigrated from southern China, where raw fish is commonly eaten and parasitism is extremely common. Those investigators who believe that liver parasitism is responsible for many cases of primary liver carcinoma point out that in a parasitized liver there is usually an associated cirrhosis as the result of mechanical irritation and cytotoxic influences by the parasite.

On the other hand, others [29,46,62] believe there is no such direct relationship between parasitism and primary liver carcinoma. Berman [5] pointed out that in the Egyptian Nile population, where 70% to 90% of the people have schistosomiasis, primary liver cancer is exceptionally rare. Also, while Schistosoma infestation is prevalent in the white races of East Africa and South Africa, primary carcinoma of the liver is rare. No conclusive statistics are available on the incidence of primary liver cancer among Orientals born in the United States, but it would appear that the incidence is no higher than among the North American white people.

Many cases of cirrhosis are without doubt the direct result of liver parasitism. Perhaps the cases of primary liver carcinoma found in association with liver parasites is due to malnutrition and other unexplained factors instead of the carcinogenic action of the parasite.

Chronic Irritation.—The liver is notorious in its ability to repair itself after injury. Occasionally, the healing process proceeds to actual overgrowth, or hyperplasia, and possibly to neoplasia. Liver cancers have been reported in association with hepatoliths,[50] virus hepatitis,[53] protozoa, and helminths.[2] Whether chronic cholestasis and infection have any relation to primary liver carcinoma is not known. Stewart, Cantarow, and Morgan [57] have demonstrated papillary and adenomatous proliferation of the bile duct mucosa with mitotic figures and periductular fibrosis as the result of chronic cholestasis.

Congenital Defects and Hereditary Factors.—Primary liver cancers appear in certain strains of laboratory mice and rats even though these animals are fed well-balanced diets and protected from stimuli which might conceivably harm the liver. It would seem safe to conclude that these animals carry an inborn trait which predisposes many of them to liver cancer. There is little evidence to conclude that such may be the case in adult humans.

As was pointed out, primary liver cancers in children do not fit the pattern of primary liver cancers in adults. There is rarely an associated cirrhosis. Since the tumor may appear in infants, it would appear that the carcinoma results from influences acting on the liver prior to birth. Wells,[66] in his study, concludes that some of the liver cancers are truly congenital.

Hemochromotosis.—Berk and Lieber [3] found primary liver carcinomas in associa-

tion with hemochromototic livers to be in the neighborhood of 7.5%. When compared to the usual incidence of 4.5% of hepatomas in simple portal cirrhosis, this seems very significant. There are others [46,50] who believe that primary carcinoma occurs no more frequently in pigmentary cirrhosis than in portal cirrhosis.

Trauma.—Berman [4] and Crawford [12] have reported cases of primary liver carcinoma which supposedly followed abdominal trauma. Since trauma to the abdomen is such a frequent occurrence, the history of abdominal trauma in a patient with primary liver cancer would not be unusual.

Clinical Features

The early symptoms of primary liver carcinoma tend to be vague, and the patient is apt to overlook them. It is difficult to separate the symptoms of early liver carcinoma from gallbladder disease, or upper gastrointestinal dysfunction. Abdominal pain is the commonest symptom. It is usually located in the epigastrium or right hypochondrium and is described as dull and aching with no relation to meals. Occasionally, however, the patient will complain of episodic pain following the ingestion of certain foods.

Weakness, weight loss, and fatigue are experienced relatively early by many patients. As long as liver function remains normal, these symptoms are minimal, but as the liver fails, the patient is apt to become rapidly cachectic. Nausea, vomiting, and constipation are associated symptoms.

In many instances, the major symptoms are due primarily to the accompanying cirrhosis. Hematemesis and melena may be noted, which is the result of ruptured esophageal varices. Ascites, dilated veins over the abdomen, hemorrhoids, splenomegaly, and other findings indicative of portal hypertension may be found. Ascites is found, either initially or terminally, in about three-fourths of the patients. It may be rapidly accumulating, necessitating frequent paracenteses for comfort. The ascitic fluid is frequently sanguinous, which is significant, since it may represent a ruptured tumor nodule on the surface of the liver, as well as a prothrombin deficiency.

Dyspnea is a late symptom, which may be due to lung metastases, to anemia, or to poor ventilation of the lungs when there is a large liver with ascites.

Several cases have been reported [1,12,41] in which the cardinal symptom was hypoglycemia, manifested by hunger, nervousness, visual disturbances, and shock.

Fever is an inconsistent finding. There may be a low-grade fever with septic type spiking above the usual elevated plateau. This is probably due to central necrosis of a large tumor nodule. Sudden, high temperature elevation frequently heralds the death of the patient.

Jaundice is seen in slightly more than half of the patients. The liver may be completely riddled with tumor nodules, and yet there will be no clinical jaundice. When jaundice is present, it is usually mild, and only rarely is deep jaundice seen.

The most significant physical finding is hepatomegaly. The liver is enlarged in three-fourths of the cases, but since it tends to extend upward, it is palpable in only about half the cases. Typically, the liver is only slightly tender and may be fixed. One may be able to palpate stony-hard nodules on the surface of the liver. The spleen may also be palpable.

The available laboratory tests are of little value in making a diagnosis of primary carcinoma of the liver. There is almost always an anemia of mild degree unless there has been actual hemorrhage, in which case the anemia may be profound. Varying degrees of leukocytosis may be noted.

Tests which measure liver-cell function remain normal surprisingly late in the disease, providing the liver is able to compensate for the malignant destruction of the parenchyma. This, of course, is not true if cirrhosis is present. The erythrocyte sedimentation rate tends to be elevated and will increase as new metastases develop.

An elevated serum alkaline phosphatase, in the absence of demonstrable bone disease, is thought by some [13,27] to be highly significant. This is indicative of obstructive disease of the liver.

Stein [54] believes that, in the presence of jaundice, the Takata-Ara reaction is useful in differentiating intrahepatic carcinoma from extrahepatic carcinoma of the bile ducts and pancreas. In his experience, the reaction is negative in carcinoma of the extrahepatic bile ducts or pancreas without hepatic metastases and positive with extensive primary carcinoma of the liver.

An elevated serum bilirubin is usually found late in the disease even in the absence of clinical jaundice. The icterus index rarely exceeds 100 units.

Diagnosis

A roentgenographic study of the liver may show hepatic enlargement, elevation and fixation of the diaphragm, esophageal varices, or localized indentations of the diaphragm, all of which make one suspicious of primary liver carcinoma. Hermosilla Diaz and Sotomayor [14] believe an important diagnostic sign in primary liver carcinoma is the elevation of the diaphragm without an adjacent pleural reaction, as is seen in subphrenic abscess. An x-ray showing the displacement of neighboring viscera when filled with contrast media may be of value. Visualization of the liver by thorium dioxide may show tumor nodules as rounded areas of reduced density. This procedure is considered to be unwise, however. A chest roentgenogram should be obtained to rule out pulmonary metastases.

Aspiration of ascitic fluid with centrifugation and examination by the Papanicolaou technique may reveal malignant cells.

Peritoneoscopy may be of value, since it allows one direct visualization of the liver. This is helpful in differentiating hepatic cirrhosis from hepatic cancer. A group at the Mayo Clinic was able to diagnose accurately 93% of hepatic malignancies by peritoneoscopic examination.[7]

The only positive method of making a diagnosis of primary carcinoma of the liver is by histologic examination of tissue excised from the liver. Several techniques for punch biopsy have been developed, but all depend on a certain element of luck in impaling a tumor nodule on the biopsy needle, and each is fraught with the danger of hemorrhage.

With present-day methods of anesthesia and whole-blood replacement, exploratory laparotomy carries very little risk and allows the surgeon direct vision of the liver and aids him in estimating the degree of spread. If the patient has any chance of cure, it is at the first laparotomy, when the involved portion of the liver must be completely removed.

Prognosis

After symptoms of primary liver carcinoma develop, the disease progresses rapidly and most patients will not survive six months. Without surgical removal of the involved liver tissue, the diease is invariably fatal. Yeomans [71] reported 16 cases of primary carcinoma of the liver in which the tumor had been removed. Six of his patients were alive and well three to seven years after surgery.

Wright,[70] in 1923, reported from England the removal of a hepatoma the size of an orange from the right lobe of the liver of a man of 60. The patient was alive without recurrence 31 months later.

Turner,[63] in 1923, removed a portion of the right lobe of the liver together with the gallbladder in a boy of 13 who had a liver-cell carcinoma. The lad was well 22 months after surgery.

Wallace [65] reported 29 patients in whom he resected primary liver carcinomas. Twenty-three survived the operation, and twelve were known to be alive and well two to five years after surgery.

Since the course of this disease is known to be rapidly fatal, an attempt at resection, whenever possible, should be made. Even though the tumor is incompletely removed,

some excellent palliative results have been obtained.

Report of Case

A 29-year-old housewife, was admitted to the Jefferson Hospital on Oct. 16, 1957, complaining of sudden, severe pain in the right upper abdominal quadrant and right lower chest. In November, 1956, she became pregnant and remained relatively asymptomatic throughout her pregnancy but did describe episodes of vague discomfort in the right hypochondrium which were unrelated to foods.

On Aug. 29, 1957, she was delivered of a normal boy. Six weeks prior to admission, she mentioned having pain in the right upper abdominal quadrant. On the night of admission, she was awakened by excruciating abdominal pain and sent to the hospital by her family physician.

On admission, all pertinent physical findings were confined to the abdomen. Palpation of the abdomen revealed a large, firm mass in the right upper quadrant, which extended from the midline downward nearly to the iliac crest and around into the right flank. The mass appeared smooth, was somewhat fixed, and moved with respiration.

Laboratory studies on admission showed a white cell count of 5,650; microhematocrit 40% or 88% of normal; urinalysis normal; BSP sulfobromophthalein less than 5% retention, and prothrombin time 57% of normal. This last finding seemed unusual in the absence of severe liver dysfunction but was repeated and found to be the same.

A flat roentgenogram of the abdomen demonstrated a large soft-tissue mass which completely filled the right side of the abdomen and extended from the liver margin to the crest to the ilium. There was displacement of the hepatic flexure and proximal half of the transverse colon into the lower abdomen and pelvic area.

A simultaneous gallbladder series and an excretory urogram showed the gallbladder to be of average size and functioning normally but displaced across the midline into the left upper quadrant by the large soft-tissue mass. Both kidneys functioned normally, but there was considerable deformity of the right kidney by extrensic pressure from above (Fig. 1). An x-ray study of the barium-filled stomach and duodenum showed marked displacement of the stomach and duodenum posteriorly and to the left by the tumor (Fig. 2). Chest films demonstrated neither elevation of the diaphragm nor other abnormalities.

On the fifth hospital day, she was taken to the operating room, and, under general anesthesia, an exploratory laparotomy was undertaken. When the peritoneal cavity was entered, an enormous tumor was encountered which appeared to arise from the right lobe of the liver but was contiguous with the right kidney.

A biopsy specimen was taken for rapid examination, and this showed malignant changes characteristic of bile duct-cell carcinoma of the liver. The original right rectus incision was extended through the right diaphragm and into the right chest through the seventh intercostal space. After

Fig. 1.—A cholecystogram of an excretory urogram showing the gallbladder displaced to the left and the right kidney depressed and distorted.

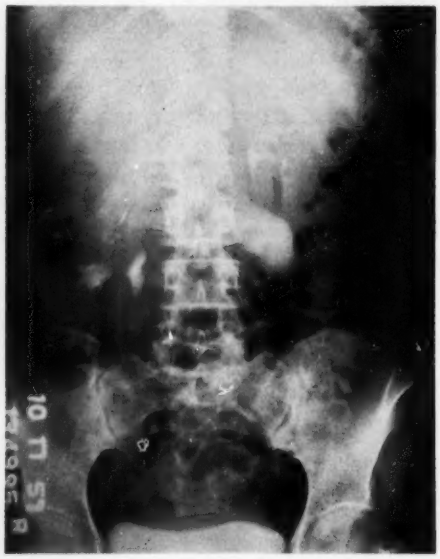

Fig. 2.—Note the large mass which fills the right upper quadrant, displacing the stomach to the left and the small bowel into the pelvis.

Fig. 3.—The right lobe of the liver, gallbladder, and right kidney removed en bloc. The right lobe weighed 2,030 gm. Note the large tumor mass, which has been incised.

this, the tumor could be visualized and its full extent estimated. The surgical attack upon the tumor necessitated a total right hepatic lobectomy together with removal of the right kidney, gallbladder and perinephritic fat en bloc. The resected lobe weighed 2,030 gm. (Fig. 3). The vena cava, left lobe of the liver, and portal triad appeared grossly to be free of the tumor, as did all the surrounding tissues.

The patient's immediate postoperative course was essentially uneventful considering the magnitude of the procedure. On the 30th postoperative day, a large subhepatic bile abscess was discovered and drained. This proved to be self-limited and she responded quickly to drainage. She did well until after Christmas, at which time she began to have upper abdominal pain and a mass was discovered in the left upper quadrant. On Dec. 30, 1957, she was explored through a left rectus incision, and the left lobe of the liver was found to be largely replaced by tumor. Numerous small nodules were seen on the serosal surfaces of the intestines, and a considerable quantity of serosanquinous ascitic fluid was aspirated.

From this time on, the patient's condition deteriorated slowly, and she finally lapsed into coma and died on Jan. 29, 1958, exactly 100 days after her original operation.

A postmortem examination was performed which showed an enormous liver filling the upper abdomen. It was not possible to determine which portion of the liver remained. The remaining liver mass weighed 2,900 gm. Metastatic tumor of the bile duct-cell type was found throughout the lungs, serosal surfaces of the bowel, and perihepatic tissues. The most unusual finding was a tumor nodule attached to the chordae tendineae of the tricuspid valve. Cardiac metastases from primary carcinoma of the liver are exceedingly rare, and, to our knowledge, such a lesion has never been reported. The remaining viscera were free of tumor. Death was thought to be due to cachexia and hepatic failure.

Treatment

The treatment of primary carcinoma of the liver consists of removing the entire lobe which contains the tumor. One may be reluctant to resect an entire hepatic lobe, particularly the right, for fear of the catastrophic hemorrhage which may develop. Actually, a lobectomy may be performed with surprisingly little blood loss if the proper plane is developed and followed.

Total right hepatic lobectomy for primary liver carcinoma is an uncommon procedure. Fineberg [18] made a careful search of the surgical literature up to March, 1955, and found only three instances of a total right hepatic lobectomy for primary liver carcinoma. To this, he added a case of his own.

The internal anatomy of the liver has been well demonstrated by Healey [30] and Goldsmith and Woodburne,[23] using liver casts made by the injection-corrosion technique. It has been shown that the interlobar fissure lies well to the right of the falciform ligament; hence, the left lobe is considerably larger than is shown in most textbooks of anatomy. The right lobe is further di-

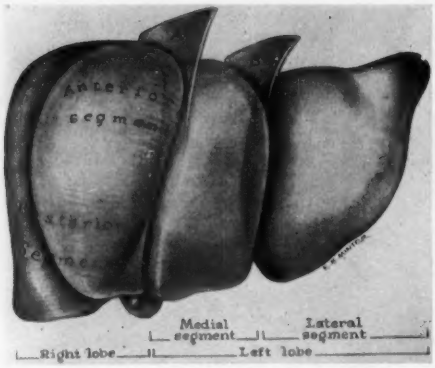

Figure 4

Fig. 4.—Diagrammatic illustration of the divisions of the liver, based upon the intrahepatic distribution of the segmentally arranged structures, i. e., the bile ducts, hepatic artery, and portal vein. *A*, lobar fissure; *B*, left segmental fissure. The right segmental fissure divides the right lobe into an anterior and a posterior segment. Reproduced with permission from Healey, J. E., Jr.: J. Internat. Coll. Surgeons 22:542, 1954; Chicago.

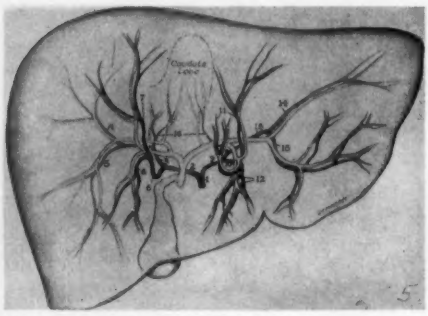

Figure 5

Fig. 5.—Composite drawing illustrating the prevailing pattern of branching of the bile ducts (light) and hepatic artery (dark). Although only the arteries are numerically indicated, the accompanying ducts carry a similar terminology: (*1*) common hepatic artery; (*2*) right hepatic artery; (*3*) posterior segment artery; (*4*) posterior-superior area artery; (*5*) posterior-inferior area artery; (*6*) anterior segment artery; (*7*) anterior-superior area artery; (*8*) anterior-inferior area artery; (*9*) left hepatic artery; (*10*) medial segment artery; (*11*) medial-superior area arteries; (*12*)medial-inferior area arteries; (*13*) lateral segment artery; (*14*) lateral-superior area artery; (*15*) lateral-inferior area artery; (*16*) caudate lobe arteries. Reproduced with permission from Healey, J. E., Jr.: J. Internat. Coll. Surgeons 22:542, 1954; Chicago.

vided into an anterior segment and a posterior segment and the left lobe into medial and lateral segments (Fig. 4). Two main ducts drain the right lobe, while there are usually three bile ducts emptying the left lobe. The hepatic artery closely follows the ramose bile duct system (Fig. 5). In the majority of liver casts studied by Healey, the portal venous system was similar to that of the hepatic artery and hepatic bile ducts in that there are two main branches from the right lobe and three from the left lobe (Fig. 6).

The hepatic veins are not segmentally distributed. Three main trunks of the hepatic vein may be distinguished: a middle

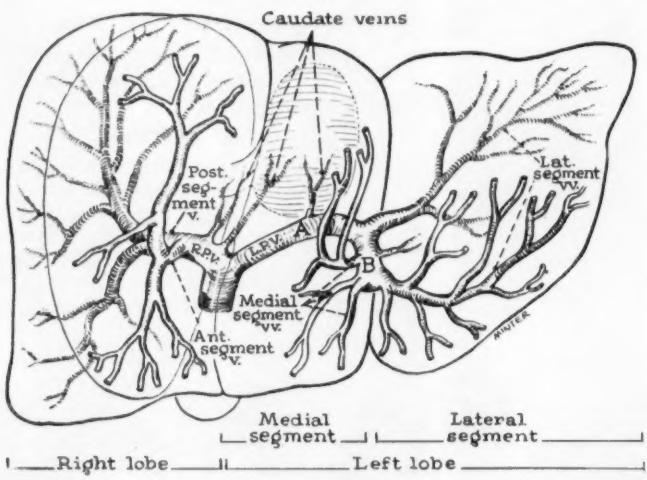

Fig. 6. — Intrahepatic distribution of the portal vein. *RPV*, right portal trunk; *LPV*, left portal trunk; *A*, pars transversus of left portal trunk; *B*, pars umbilicus of left portal trunk. Reproduced with permission from Healey, J. E., Jr.: J. Internat. Coll. Surgeons 22:542, 1954; Chicago.

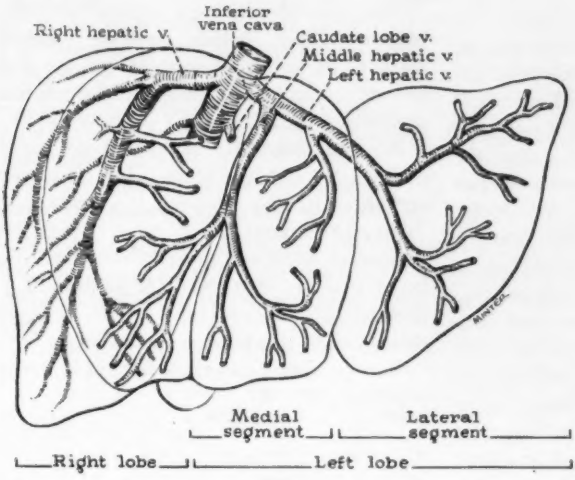

Fig. 7.—Prevailing pattern of the hepatic veins in the human liver. Reproduced with permission from Healey, J. E., Jr.: J. Internat. Coll. Surgeons 22:542, 1954; Chicago.

hepatic vein, which lies in the lobar fissure; a left hepatic vein, which lies in the upper portion of the left segmental fissure, and a right hepatic vein, which lies in the right segmental fissure[30] (Fig. 7).

Some type of thoracoabdominal incision gives the best exposure and allows the liver to be delivered into the right thorax after the hepatic ligaments have been divided.

In beginning the resection of the right lobe of the liver, one commences by ligating and dividing the cystic duct and cystic artery, leaving the gallbladder attached to the liver. The anterior edge of the liver is elevated so that the base of the liver may be visualized. The right hepatic duct, right hepatic artery, and right branch of the portal vein are ligated, transfixed, and divided.

One must then delineate the lobar fissure which lies along an imaginary line drawn from the gallbladder fossa below to the fossa of the inferior vena cava above.[18] Glisson's capsule is then incised over the lobar fissure and the parenchyma separated by blunt dissection. The intrahepatic vessels and ducts are individually clamped and ligated as they are encountered. The dissection is carried deeper into the parenchyma until the inferior vena cava is seen. The branches of the right hepatic vein are identified, ligated, and divided. Oozing from the cut surface of the liver can usually be controlled by manual compression by the first assistant, then by covering the area with gelatin surgical sponge saturated with thrombin and held in place by gentle pressure. Large mattress sutures are rarely necessary.

A soft rubber catheter is placed in the right pleural space and connected to an underwater seal. Several Penrose drains are placed in the right subphrenic and subhepatic spaces and brought to the outside. The wound is then closed in the usual manner.

In the postoperative management of the patient, one must remember that hepatic function has been suddenly reduced. In the immediate postoperative period, parenteral alimentation is best accomplished by the use of 10% invert sugar because of its nitrogen-sparing effect and since it is easily converted into liver glycogen. When normal peristalsis resumes, a high-calorie, high-protein, high-carbohydrate diet should be given. Supplementary B-complex vitamins and ascorbic acid are recommended. Drugs that are detoxified by the liver should be avoided.

Comment

Primary carcinoma of the liver has an extremely poor prognosis, but one must not adopt the attitude that a surgical attack upon such a tumor is an utterly futile procedure. It is true that by the time the tumor makes its presence known it has usually spread beyond the limits of resectability. Rarely, however, an early primary hepatic malignancy may be discovered at laparotomy which may be completely excised. It is this group that has the only chance of cure, and, should such a tumor be discovered, the entire lobe which contains it must be removed. It behooves every practitioner of abdominal surgery to familiarize himself with the internal anatomy of the liver, in order that a lobectomy of the liver may be carried out if the indication arises.

We believe the case presented here is unusual in that it occurred in a young woman whose symptoms were masked by her pregnancy. Her age and sex, histologic tumor type, rapid downhill course, widespread metastases, and peritoneal seeding are somewhat unusual findings. Cardiac metastases from primary liver carcinomas are rare, being found in about 1% of cases. In reviewing the literature, we have been unable to find an instance of cardiac metastasis similar to the one reported here.

Summary

Primary carcinoma of the liver is rare in the United States, but its world-wide incidence is much higher, particularly in Africa and the Orient.

It is primarily a disease of adults, but a number of cases have been reported in infants and children. The incidence is higher in males than in females.

Three gross tyes of primary liver carcinoma may be found: massive, nodular, or diffuse.

Primary liver carcinoma is divided into three histologic groups: liver-cell type (malignant hepatoma), bile duct-cell type (malignant cholangioma), and the atypical, or mixed, type (malignant cholangiohepatoma).

The possible etiologic agents are discussed. These include cirrhosis, dietary factors, parasites, chronic irritation, congenital defects and hereditary factors, hemachromotosis, and trauma.

No pathognomonic symptoms are noted early in the disease. Later, abdominal pain and the discovery of a mass are the most important findings.

Several diagnostic procedures which may be of help are discussed. A positive diagnosis may be made only by microscopic examination of a portion of the tumor.

The prognosis is grave but not entirely hopeless.

A case of primary carcinoma of the liver in a young woman with right hepatic lobectomy is presented.

The only treatment of primary liver carcinoma is complete surgical extirpation of the tumor. The surgical anatomy of the liver and the operative technique are discussed.

Jefferson Hospital, 1313 Franklin Rd.

REFERENCES

1. Beers, D. N., and Morton, J. J.: Primary Carcinoma of the Liver with Hypoglycemia, Am. J. Cancer 24:51, 1935.

2. Berk, J. E.: Primary Carcinoma of the Liver, Clinics 3:582, 1944.

3. Berk, J. E., and Lieber, M. M.: Primary Carcinoma of the Liver, Am. J. M. Sc. 202:708, 1941.

4. Berman, C.: Primary Carcinoma of the Liver in the Bantu Races of South Africa, South African J. M. Sc. 5:54, 1940.

5. Berman, C.: The Pathology of Primary Carcinoma of the Liver in the Bantu Races of South Africa, South African J. M. Sc. 6:11, 1941.

6. Bigelow, N. H., and Wright, A. W.: Primary Carcinoma of the Liver in Infancy and Childhood, Cancer 6:170, 1953.

7. Bondurant, R. F.: Primary Carcinoma of the Liver, Virginia M. Month. 79:305, 1952.

8. Campbell, J. G.: Spontaneous Hepatocellular and Cholangiocellular Carcinoma in the Duck, Brit. J. Cancer 3:198, 1949.

9. Charache, H.: Primary Carcinoma of the Liver: Report of a Case and Review of the Literature, Am. J. Surg. 43:96, 1939.

10. Counseller, V. S., and McIndoe, A. H.: Primary Carcinoma of the Liver, Arch. Int. Med. 37:363, 1926.

11. Cramer, W.: The Origin of Cancer in Man, J. A. M. A. 119:309, 1942.

12. Crawford, W. A.: Hypoglycemia with Coma in a Case of Primary Carcinoma of the Liver, Am. J. M. Sc. 181:496, 1931.

13. Cunningham, R. M.: Primary Carcinoma of the Liver, Bull. School Med. Univ. Maryland 28:61, 1943.

14. Hermosilla Diaz, F., and Sotomayor, M. O.: Cáncer primitivo del hígado, Rev. méd. Chile 72: 685, 1944.

15. Edmondson, H. A., and Steiner, P. E.: Primary Carcinoma of the Liver: Study of 100 Cases Among 48,900 Necropsies, Cancer 7:462, 1954.

16. Eggel, H.: Über das primäre Carcinom der Leber, Beitr. path. Anat. 30:506, 1901.

17. Ewing, J.: Neoplastic Diseases: A Treatise on Tumors, Ed. 4, Philadelphia, W. B. Saunders Company, 1940, p. 735.

18. Fineberg, C.; Goldburgh, W. P., and Tenpleton, J. Y., III: Right Hepatic Lobectomy for Primary Carcinoma of the Liver, Ann. Surg. 144: 881, 1956.

19. Fried, B. M.: Primary Carcinoma of the Liver, Am. J. M. Sc. 168:241, 1924.

20. Gardner, L. W.: Primary Carcinoma of the Liver, Harper Hosp. Bull. 1:36, 1941.

21. Gelfand, M.: Primary Carcinoma of the Liver, Clinico-Pathological Conference, New York J. Med. 47:1136, 1947.

22. Gilbert, C., and Gillman, J.: Diet and Disease in the Bantu, Science 99:398, 1944.

23. Goldsmith, N. A., and Woodburne, R. T.: The Surgical Anatomy Pertaining to Liver Resection, Surg. Gynec. & Obst. 105:310, 1957.

24. Greene, J. M.: Primary Carcinoma of the Liver: Ten Year Collective Review, Internat. Abstr. Surg. 69:231, 1939; in Surg. Gynec. & Obst., Sept., 1939.

25. Gregory, R.: Primary Carcinoma of the Liver: Tumor Thrombosis of the Inferior Vena Cava and Right Auricle, Arch. Int. Med. 64:566, 1939.

26. Gustafson, E. G.: An Analysis of 62 Cases of Primary Carcinoma of the Liver Based on 24,400 Necropsies at Bellevue Hospital, Ann. Int. Med. 11:889, 1937.

27. Gutman, A. B.; Olson, K. B.; Gutman, E. B., and Flood, C. A.: Effect of Disease of the Liver and Biliary Tract upon Phosphatase Activity of Serum, J. Clin. Invest. 19:129, 1940.

28. Gyorgy, P.; Poling, C. E., and Goldblatt, H.: Necrosis, Cirrhosis and Cancer of the Liver in Rats Fed a Diet Containing Dimethylaminoazobenzene, Proc. Soc. Exper. Biol. & Med. 47:41, 1941.

29. Hartz, P. H.: The Role of Schistosomiasis in the Etiology of Cancer of Liver in the Chinese, Arch. Path. 39:1, 1945.

30. Healey, J. E., Jr.: Clinical Anatomic Aspects of Radical Hepatic Surgery, J. Internat. Coll. Surgeons 22:542, 1954.

31. Herxheimer, G.: Leber Gewachse, in Handbuch der speziellen pathologischen Anatomie und Histologie, edited by F. Henke and O. Lubarsch, Berlin, Springer-Verlag, 1930, Vol. 5, p. 797.

32. Hoyne, R. M., and Kernohan, J. W.: Primary Carcinoma of the Liver: A Study of 31 Cases, Arch. Int. Med. 79:532, 1947.

33. Hudimaki, S., cited by Charache.[9]

34. Kelsch, A., and Kiener, P-L.: Contribution à l'histoire de l'adénome du foie, Arch. physiol. norm. et path. 3:622, 1879.

35. Kennaway, E. L.: Cancer of the Liver in the Negro in Africa and in America, Cancer Res. 4:571, 1944.

36. Lisa, J. R.; Solomon, C., and Gordon, E. J.: Secondary Carcinoma in Cirrhosis of the Liver, Am. J. Path. 18:137, 1942.

37. Loesch, J.: Primary Carcinoma of the Liver, Arch. Path. 28:223, 1939.

38. Lynch, K. M.: Primary Liver Carcinoma: Relation to Yellow Atrophy Cirrhosis, South. Med. J. 30:1043, 1937.

39. Matthews, W. R.: Primary Carcinoma of the Liver, Tri-State M. J. 10:2511, 1940.

40. Miller, J. A.; Miner, D. L.; Rusch, H. P., and Baumann, C. A.: Diet and Hepatic Tumor Formation, Cancer Res. 1:699, 1941.

41. Nadler, W. H., and Wolfer, J. A.: Hepatogenic Hypoglycemia Associated with Primary Liver Cell Carcinoma, Arch. Int. Med. 44:700, 1929.

42. Neumann, M. A.: Primary Carcinoma of the Liver with Metastasis to Bone: Report of a Case, Am. J. Path. 20:895, 1944.

43. Noeggerath, E.: Geburtshindernis in folge eines lebercarcinomas beim neugeboren, Deutsche Klin. 6:496, 1854.

44. Opie, E. L.: The Influence of Diet on the Production of Tumors of the Liver by Butter Yellow, J. Exper. Med. 80:219, 1944.

45. von Rokitansky, C.: A Manual of Pathological Anatomy, translated from German Ed. 3 by E. Sieveking, Philadelphia, Blanchard and Lea, 1855, Vol. 2.

46. Rosenberg, D. M. L., and Ochsner, A.: Primary Carcinoma of the Liver: An Analysis of 55 Autopsied Cases, the Record of a Case with Resection, and a Review of the Recent Literature, Surgery 24:1036, 1948.

47. Rowen, H. S., and Mallory, F. B.: A Multinucleated Liver-Cell Carcinoma, Am. J. Path. 1:677, 1925.

48. Rusch, H. P., and Brumann, C. A.: Nutritional Aspects of the Cancer Problem, Nutrition Abstr. & Rev. 4:353, 1946.

49. Sabourin, C.: Contribution a l'etude des lesions du parenchyme hepatique dans la cirrhose; essai sur l'adenome du foie, Thesis, Paris, No. 39, 1881.

50. Sanes, S., and MacCallum, J. D.: Primary Carcinoma of the Liver: Cholangioma in Hepatolithiasis, Am. J. Path. 18:675, 1942.

51. Schatzki, R.: Roentgenological Diagnosis of Primary Carcinoma of the Liver, Am. J. Roentgenol. 46:476, 1941.

52. Sheldon, J. H.: Haemochromatosis, London, Oxford University Press, 1935.

53. Sheldon, W. H., and James, D. F.: Cirrhosis Following Infectious Hepatitis: A Report of 5 Cases, in 2 of Which There Was Superimposed Primary Liver Cell Carcinoma, Arch. Int. Med. 81:666, 1948.

54. Stein, H. B.: The Takata-Ara Reaction in Jaundice, South African J. M. Sc. 9:41, 1944.

55. Steiner, M. M.: Primary Carcinoma of the Liver in Childhood: Report of 2 Cases, with Critical Review of the Literature, Am. J. Dis. Child. 55:807, 1938.

56. Steiner, P. E.: Incidence of Carcinogenic Factors in Livers of Cancer, Non-Cancer, Cirrhotic, and Negro Patients, Cancer Res. 3:385, 1943.

57. Stewart, H. L.; Cantarow, A., and Morgan, D. R.: Changes in the Liver of the Cat Following Ligation of Single Hepatic Ducts, Arch. Path. 23:641, 1937.

58. Strong, G. F., and Pitts, H. H.: Further Observations on Primary Carcinoma of the Liver in Chinese, Ann. Int. Med. 6:485, 1932.

59. Strong, G. F.; Pitts, H. H., and McPhee, J. G.: Primary Carcinoma of the Liver—a 25 Year Study, Ann. Int. Med. 30:791, 1949.

60. Thompson, W. T., Jr., and Higgins, W. H., Jr.: Primary Carcinoma of the Liver, Virginia M. Month. 79:305, 1952.

61. Tomlinson, W. J., and Wolff, E.: Primary Liver-Cell Carcinoma in Infancy: Report of Two Cases, One Showing Calcification, Am. J. Clin. Path. 12:321, 1942.

62. Tull, T. C.: Primary Carcinoma of the Liver: A Study of 134 Cases, J. Path. & Bact. 35:557, 1932.

63. Turner, G. G.: A Case in Which an Adenoma Weighing 2 Pounds and 3 Ounces Was Successfully Removed from the Liver, with Remarks on Subject of Partial Hepatectomy, Proc. Roy. Soc. Med. (Sect. Surg.) 16:43, 1923.

64. Usuda, H., and Uchida, K.: Pathological Study of Primary Carcinoma of the Liver in Formosa and the Far East, Science Bull. 4:1, 1944.

65. Wallace, R. H.: Resection of the Liver for Hepatoma, Arch. Surg. 43:14, 1941.

66. Wells, H. G.: The Occurrence and Significance of Congenital Malignant Neoplasms, Arch. Path. 30:535, 1940.

67. Widerhofer, cited by Steiner, M. M.[66]

68. Wilbur, D. L.; Wood, D. A., and Willett, F. M.: Primary Carcinoma of the Liver, Ann. Int. Med. 20:453, 1944.

69. Winer, J. H., and Schroeder, C. R.: Malignant Hepatoma in the White-Tailed Deer (Odocoileus Osceola), Am. J. Cancer 38:268, 1940.

70. Wright, G.: Primary Carcinoma of the Liver Excised by Operation, Proc. Roy. Soc. Med. 16:56, 1923.

71. Yeomans, F. C.: Primary Carcinoma of the Liver: Operation for Recurrence over 7 Years After Primary Operation, J. A. M. A. 64:1301, 1915.

72. Yoshida, T.: Studien über die Entwicklung des experimentellen Hepatoms durch o-Amidoazotoluol, besonders die stufenweise Verfolgung der Leberveranderung bis zur Carcinomentstehung, Tr. Jap. Path. Soc. 24:523, 1934.

The Thyroid Nodule

A Study of Its Significance As Related to Its Size and Known Duration

G. C. FREEMAN, M.D., Honolulu

Is the routine removal of all thyroid nodules justified? Is the size of a thyroid nodule significant? Are multiple nodules unlikely to mean carcinoma? Is the duration of the nodule of importance? Should the fact that an unsuspected lesion is found on routine examination influence the advice to the patient? Answers to these questions have been looked for in 155 lesions operated upon by me and 220 removed by the other members of the Surgical Department of the Straub Clinic since 1947.

Terminology

In this report, the term adenoma is meant to signify epithelial neoplasm as distinguished from a non-neoplastic thyroid nodule. In some instances this distinction is difficult, but it has been based on both the gross appearance of the lesion and the usual microscopic features that distinguish a true adenoma (Shields Warren [5]).

Table 1 classifies histologically these 375 nodular thyroids. One hundred six were characteristic of nontoxic nodular goiter. Some form of chronic thyroiditis was the predominant lesion in 46 patients.

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Department of Surgery, Straub Clinic.

TABLE 1.—*Classification of 375 Nodular Thyroid Glands*

Non-neoplastic nodules		
Adenomatous goiter	106	
Thyroiditis	46	
Total	152 (40%) *	
Neoplastic nodules		
Adenomas	160	
Carcinomas	55	(16.8%)
Potentially malignant	8	
Total	223 (60%)	

* Figures in parenthesis represent per cent of total in series.

The remaining 223, almost 60% of the total, had some type of neoplasm. One hundred sixty of these neoplasms were adenoma, customarily referred to as the follicular type with no attempt made to divide them further. The remaining 63 neoplasms included 55 frank carcinomas and 8 lesions regarded as borderline or potentially malignant. These borderline lesions include four of the nonencapsulated sclerosing tumors thought by Graham [2] to be benign but more recently reported by Klinck and Winship [3] to be able to metastasize. The other four potentially malignant lesions were well-encapsulated papillary cystadenomas with no evidence of invasion. These 63 potential or actual malignancies comprise 16.8% of the nodular thyroids removed.

These figures are comparable to those obtained in the routine removal of all breast nodules. Strode [4] recently reported a series of 1,080 breast nodules removed with the same 40 to 60 distribution between some form of fibrocystic disease and the neoplasms. In the entire series there were 220 malignant tumors, 20% of the total. These figures almost exactly parallel the experience with thyroid nodules.

Multiple Colloid Adenomatous Goiter

Nontoxic nodular goiter is the term generally used to describe the changes in the thyroid gland which are found typically in the endemic goiter belts of the world. I have preferred the term urged by Shields Warren [5]—a mouthful, to be sure, but an accurate descriptive title, namely, multiple

colloid adenomatous goiter (MCAG) also shortened to adenomatous goiter.

This condition was the most important pathological change in 106 of the patients in this series. In two patients, carcinoma was superimposed upon these changes. One was a 57-year-old Filipina, native of her islands, who had had a goiter for 25 years, which had undergone recent enlargement and which upon resection was found to contain extensive alveolar adenocarcinoma. In the second case with MCAG, a nonencapsulated sclerosing tumor was noted as an incidental pathological finding.

The fact that 106 out of 375 patients were found to have this lesion does not make Hawaii an endemic goiter belt, but emphasizes that not all nodular thyroids are neoplasms even in a community such as ours, an island surrounded by the vast Pacific where iodine in the environment is plentiful. Sixty-four of these adenomatous goiters were found in Caucasians, many of them natives of the mainland United States who had moved to Hawaii. However, MCAG was diagnosed in 13 Japanese, 14 Polynesians, 4 Filipinos, and 7 Chinese. In all of these latter groups, fish is a staple item in the diet, and it would appear that iodine deficiency was not the mechanism at work in the production of their disease. At least 40 of this group of patients with MCAG had frank goiters whose size and symptoms were considered to be the prime indication for their surgical removal, with the thought of cancer prophylaxis a secondary consideration. Many of the remaining 66 who were found to have MCAG were suspected of harboring this entity; however, the palpable changes were not diagnostic, and, in accord with our established policy of removing all nodules in the thyroid, they were subjected to surgery.

Thyroiditis

The incidence of chronic thyroiditis in this series is high, with 46 patients showing some form of this condition as the major pathological finding. Hashimoto's struma was the commonest type found, but also noted were Riedel's struma and chronic granulomatous thyroiditis, sometimes called DeQuervain's thyroiditis. A number of thyroids with other entities considered more significant also showed chronic thyroiditis. One patient with a full-blown Hashimoto's struma was also found to harbor a papillary adenocarcinoma.

Most of these patients with thyroiditis probably would be best not operated upon if the clinician could be sure of the diagnosis. Many times, however, the surgeon sees a patient whose thyroid presents a diffuse firm or hard enlargement, sometimes bilateral, but also sometimes of only one lobe, where it is impossible to rule out the presence of carcinoma. We also have observed patients with the typical clinical picture of a Hashimoto's struma and have deferred surgery only to see the gland over a period of months or years develop a nodular change that made it impossible to rule out malignancy, an association which has been emphasized by Dailey et al.[1]

In an attempt to avoid operating upon patients with thyroiditis, I have recently adopted a more conservative attitude. When findings suggest diffuse thyroiditis and no nodule is felt in the thyroid, needle aspiration biopsy has been done, and, if the clinical impression is confirmed, the patient is merely kept under observation. If the lesion increases in size or becomes nodular, thyroidectomy is advised. To date I have not had cause to regret this approach.

Neoplasms

In this series of 375 nodular thyroids, 223 (60%) were neoplastic, and 63 of the neoplasms were either preinvasive forms of malignancy or actual carcinoma. This is certainly comparable in prophylactic value to the well-accepted practice of the routine removal of all breast nodules.

The Incidental Nodule

The importance of the routine physical examination is being stressed in many quar-

ters with emphasis placed on the early recognition and treatment of cancer. In this series of 375 nodular thyroids operated upon, 164 of the nodules were picked up on routine examination. In most instances the patient had been completely unaware of the nodule, or, in a few cases, had ignored it because of apathy or previous advice that it was nothing of consequence. Of these 164 nodules, 70 were adenomas and 29 were carcinomas; almost half of the total number of malignant lesions in the whole series were previously unsuspected by the patient.

Duration of the Nodule

If the concept that benign lesions of the thyroid undergo malignant change were correct, it would be probable that the greater the duration of the nodule the greater would be the probability of its being malignant. On the assumption that the lesions that were discovered on routine examination were on the average of shorter duration than those noted by the patient, these two groups were compared. Of those found on routine examination, 17% were malignant; 15% of the 211 complained of were cancerous. Elimination of the non-neoplastic nodules does not change these ratios much. Of 99 neoplastic nodules discovered on routine examination, 29 (30%) were malignant (Table 2). Of the 126 neoplastic nodules first noted by the patient anywhere from one day to 10 years before the surgery, 34 were malignant (27%). Of neoplastic nodules present for more than a year, 15 of 40 lesions (38%) were malignant. It would be unwise to conclude that the older nodules are more likely to be malignant. However, the corollary seems to be a safe statement: just because a nodule is of recent origin is no assurance that it is benign.

TABLE 2.—*Duration of the Neoplastic Nodule*

Duration	Adenoma	Carcinoma	Total
Incidental discovery	70	29 (30%) *	99
Less than one month	25	6 (19%)	31
One month to one year	23	11 (32%)	34
Over one year	25	15 (38%)	40
Not stated	17	2	19
Subtotal	90	34 (27%)	124
Composite total	160	63 (28%)	223

* Figures in parenthesis represent per cent of line total.

Size of the Nodule

Table 3 demonstrates that the size of the thyroid lesion is no criterion by which to determine its benignancy or malignancy, or even of its invasiveness when shown to be malignant. These figures are based on the 60 adenomas and 30 carcinomas personally observed. The majority of the lesions (whether benign or malignant) were 2 cm. in diameter or less, with a ratio of one carcinoma for every two adenomas regardless of the size of the nodule found.

Also, the incidence of a discovery of metastatic nodes, either clinically or at surgery, was not related to the size of the lesion. Exactly 50% of the lesions below 2 cm. in size showed metastasis and the same was true for those larger than 2 cm.

Multiple Nodules

Many series of surgically treated nodular thyroids distinguish between the gland with a single nodule and that with multiple. In our practice at the Straub Clinic, we have not been influenced by this observation in our recommendation to the patient and have advised removal of all nodules. Most of the thyroids with solitary nodules proved to be neoplasms, but a number were found at surgery actually to contain multiple nod-

TABLE 3.—*Size of the Nodule*

Maximum Diameter, Cm.	No. of Adenomas	No. of Carcinomas	No. with Pos. Nodes
1.0	9	4	3
2.0	28	12	5 (1 bilateral)
3.0	10	6	3 (1 bilateral)
>3.0	13	5	2 (1 bilateral)
Not stated		3	2 (1 bilateral)
	—	—	—
Total	60	30	15

ules of adenomatous goiter. Most of the thyroids thought to have changes characteristic of MCAG were found to contain this lesion. It is of great significance, however, that eight carcinomas presented with more than one nodule; one, already referred to, was an obvious MCAG of long duration which was found to have undergone malignant change, and another was an obvious, rapidly growing undifferentiated carcinoma (the only one in the whole series). The remaining six were small multinodular glands which were found to contain carcinoma and it would have been very easy on clinical grounds to have considered these to be small multinodular adenomatous goiters. Certainly, the patient's best interest would not have been served by delay in these instances.

Illustrative Cases

CASE 1.—A 19-year-old Chinese-Hawaiian mother of one child came to the office the day after she discovered a nodule in the left side of her neck. A firm, discrete, 2 cm. mass was felt in the left lobe of the thyroid, without evidence of adenopathy. At surgery, the lesion was a papillary carcinoma; regional nodes were involved, and a left radical neck dissection was done. The nodes contained metastatic carcinoma.

CASE 2.—A 27-year-old pregnant Caucasian woman was found on prenatal examination to have a 1.5 cm. nodule in the right lobe of the thyroid and a second small nodule on the left. Both nodules were removed, the former by a right lobectomy. This was examined by frozen section and showed a follicular structure. The other nodule was a normal lymph node, but in the lower pole of the left lobe of the gland was a second hard, flat 1 cm. nodule, which was removed and found to be a papillary adenocarcinoma. Further search of the neck then revealed bilateral involvement of the paratracheal lymph nodes. A radical neck dissection was done on the left, the site of the papillary lesion, and half of the resected nodes showed metastasis. Six weeks later, a right radical neck dissection was done and 3 of 16 resected nodes were involved.

Both of these cases illustrate to what extent even this "favorable" type of malignancy of the thyroid can have spread when treated at the time they are first observed. In addition, the second case illustrates that even small multinodular glands are by no means innocent.

CASE 3.—A 26-year-old, single, Caucasian nurse, was found in a routine employment examination by a hospital resident to have a 1 cm. nodule in the left lobe of the thyroid. At this, she volunteered the information that her doctor at home had noted the same nodule six months previously but had reassured her that it was nothing to be concerned about. At operation, two nodules were found, one in each lobe. The one on the left was found to be a papillary carcinoma. A left radical neck dissection was done in addition to a total thyroidectomy. One node contained metastatic carcinoma, and further examination of the thyroid nodules revealed them to be a mixed papillary and follicular adenocarcinoma.

This case illustrates the danger of compromising when dealing with thyroid nodules and again that even the presence of multiple nodules is no assurance of benignancy. The size of the lesion is also no assurance of safety.

Age and Sex

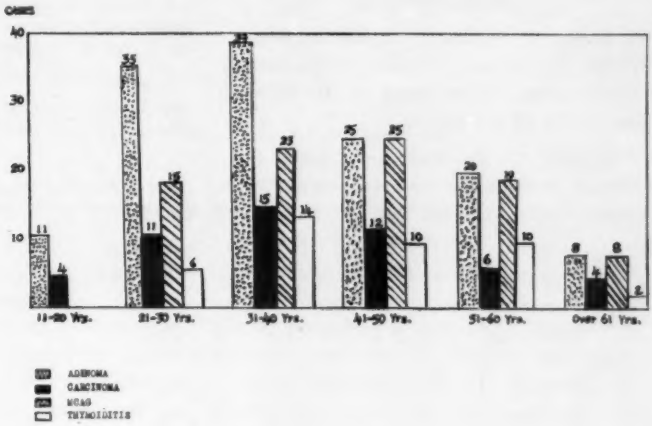
It has been reported by many authors that the age and sex of the patient are important in predicting whether a given nodule is likely to be benign or malignant. It is generally agreed that the solitary nodule in the male patient has the greatest likelihood of being malignant. The young female with a solitary nodule is also said to have a greater chance of harboring a malignancy than a middle-aged female.

In this series of 375 cases, there were 325 females and 50 males. In the female patients, 58% of the lesions were neoplastic

TABLE 4.—*Sex of the Patient*

	325 Females	50 Males
Non-neoplastic		
M. C. A. G.	93	13
Thyroiditis	42	4
Total	135	17
Neoplastic		
Benign	138	22
Malignant	52 (16%) *	11 (22%)
Total	190 (58%)	33 (66%)

* Figures in parenthesis represent per cent of column total.



Incidence of lesions according to age.

and 16% were malignant; in the males, 66% were neoplastic and 22% were malignant. It can be seen then that there is little to choose on the basis of the sex of the patient (Table 4).

In the Chart the influence of age is studied. For this purpose, male patients have not been included because of their small number and because it is generally accepted that all nodules in male patients should be removed. The Chart indicates that the distribution of lesions is not significantly different regardless of the age group. It does show that the peak incidence of adenomas is in the fourth decade and that of adenomatous goiter is in the fifth decade.

Summary

A series of 375 nodular thyroids has been studied in an attempt to learn if there are any clinical features that will reliably indicate the nature of the lesion. Of all of the lesions that were removed 60% were neoplastic. Over 16% were frankly or potentially malignant. Certainly, these figures justify the policy of routine removal of all thyroid nodules.

There were a large number of patients operated upon in whom the nodule was an incidental finding in a routine physical examination. Of 164 such patients, 70 were found to have adenomas and 29, carcinomas. Patients who had been aware of their lesions showed approximately the same ratio of benignancy to malignancy.

In those cases where sufficient data were available, it was found that more than half of the neoplastic nodules were 2 cm. or less in size. The ratio of benign to malignant lesions was not significantly influenced by size.

The percentage of malignancy in the males is somewhat higher than in females, but the difference is not great. There is a significant percentage of neoplasms, both benign and malignant, regardless of the age group of the patient.

Conclusions

1. The routine removal of all thyroid nodules is justified by the high incidence of carcinoma in such nodules.

2. The age and sex of the patient, the size or duration of the nodule, and even multiplicity of nodules are not reliable criteria for determining the likelihood of carcinoma being present.

3. Careful physical examination will reveal a large number of unsuspected thyroid nodules in which the incidence of carcinoma is the same as in those that were clinically apparent to the patient.

Kapiolani St. at Thomas Sq. (14).

REFERENCES

1. Dailey, M. E.; Lindsay, S., and Skahen, R.: Relation of Thyroid Neoplasm to Hashimoto's Disease of the Thyroid Gland, A. M. A. Arch. Surg. 70:291 (Feb.) 1955.

2. Graham, A.: The Malignant Thyroid, Proc. Interstate Postgraduate Medical Assembly North America (1927) 3:264-269, 1928.

3. Klinck, G. H., and Winship, T. A.: Occult Sclerosing Carcinoma of the Thyroid, Tr. American Goiter A. 1955, pp. 287-295.

4. Strode, J. E.: Tumors of the Breast Occurring in Hawaii, Ann. Surg. 144:872-880 (Nov.) 1956.

5. Warren, S.: The Classification of Tumors of the Thyroid, Am. J. Roentgenol. 46:447-450 (Oct.) 1940.

The Splenic Artery

Its Special Anatomy in Reference to Use as a Vascular Graft

ROBERT I. LOWENBERG, M.D., New Haven, Conn.
With the Technical Assistance of Anthony Piccirillo and James Norn

The surgery of the circulatory system has reached farther and farther into the vital structures of the body. Attempts have been made to operate on most parts of the human body. One of the limitations of concern to vascular surgery is the problem of reestablishing arterial continuity. Congenital, traumatic, and obliterative vascular lesions sometimes involve lengths of artery which cannot be readily bridged. In an attempt to fill such gaps, vascular tissues from adjacent, as well as distant, parts of the same or different bodies have been used. Segments of autologous vein are most readily available donor tissue, although they may not be the most desirable. Homologous and heterologous preserved arterial and venous segments are being used in some centers where a blood-vessel bank is available. These have been used with some success, although their supply is presently limited. Peritoneum-covered fascia has been used as vascular substitute and for vessel patching.[5] Foreign materials also have been used to complete vascular anastomoses. During the last war, Vitallium tubes were used by Blakemore and his colleagues.[2] A similar technique has been recently used in conjunction with a cloth-fabric prosthesis. Tubes of tantalum have been employed by Weiss and Lam.[28] As long ago as 1900, magnesium was used by Payr in an ingenious device as a vascular bridge.[21] During healing, the metallic substance was absorbed by the body juices. Carrel,[4] in 1902, and Tuffier with Carrel,[26] in 1914, as well as others, have used glass tubes to reestablish vascular flow, but this method has its chief use as providing a temporary shunt. The extensive literature on this subject may be reviewed elsewhere.[7,22] The methods designed for securing and utilizing the vascular grafts are too numerous to list at this time. Suffice it to state that satisfactory techniques are now fairly well established and widely used; each school has set up its own criteria and technique for accomplishing the desired end-result.

Most of the established methods for vascular grafting originated in the laboratory. Many methods have been studied, most of which are not yet suitable for clinical trial. Homologous tissues have been used extensively, grafting them into adult,[3,8,29] as well as adolescent animals.[17] All manner of techniques for preservation have been attempted, and many papers have been written on the subject.[13,23,29] Heterologous tissues have been given a more limited trial. Since many unknown factors exist, the fate of many types of grafts is uncertain, and long-term results are the key to the problem.

This being so, it becomes important to seek additional alternative methods. I have under study the use of ureter to bridge arterial defects.[16] Recent long-term studies conducted by Touroff and Nabatoff,[25] using venous grafts in dog aortas, show discouraging results at the end of 24 and 30 months. Aortograms performed on these animals show slow but progressive aneurysmal dilatation of the venous graft. Should this continue for another 12 to 24 months, all of the complications of an aneurysm must

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be anticipated. Similar findings have been described by Johnson [15] and others in short-term observations.

Any artery in the body which could be sacrificed and used for arterial grafting might be life-, organ-, or limb-saving. No one can question the logic of using the same type of tissue as that which must be replaced, namely, autologous arterial tissue. Moreover, experience has taught us that this tissue has the greatest yield in terms of good results. The splenic artery seems to fit this requirement best.

According to Cunningham,[6] the splenic artery is the largest branch of the celiac artery.

Accompanied by the splenic vein, which lies below it, it runs a tortuous course behind the stomach and the lesser sac of peritoneum along the upper border of the pancreas. It lies in front of the left suprarenal gland and the upper part of the left kidney, and passes forward between the two layers of the lieno-renal ligament, in which it divides into five to eight terminal splenic branches which enter the hilum of the spleen and supply the splenic substance.

There are pancreatic, short gastric, and omental branches, as well as the left gastroepiploic artery, which commonly arises from the ventral surface of the splenic artery. Rarely, the splenic artery may have an anomalous origin from the aorta directly, or from the middle colic, the left hepatic, or the superior or inferior mesenteric artery.

The relationship of splenic artery to splenic vein is of considerable significance. Gerber [10] found that in about 54% of 55 dissections, the artery was anterior to the vein. In less than 2% the artery was posterior to the vein. In the remainder, or about 44% of his dissections, the splenic artery was found to be wound about the vein. Whereas the vein took a straight course from hilus to portal vein, the splenic artery wound tortuously from celiac axis to splenic hilus. Michels,[18] in an excellent study of a large number of specimens, made many anatomical observations which would be of great value to anyone interested in the surgical applications of this concept. Woodburne,[30] in his studies of the arterial supply to the pancreas, demonstrated the branches of the splenic artery. The pancreatica magna, a branch of the splenic, was found in 64.7% of dissections.

The splenic artery, by virtue of its length and caliber and its availability through splenectomy, may be used either in continuity or as a free arterial graft. A study was undertaken to determine the surgically important features of the splenic artery, with particular reference to its suitability for arterial grafting. At the time this study was initiated, no knowledge was at hand that the splenic artery had ever been used in this manner clinically or that it had been considered for this type of surgery; information concerning this came to light when the laboratory part of this study was nearing completion. It has been known for some time that the internal mammary artery was being transplanted into the myocardium by Vineberg,[27] with encouraging results in a limited number of cases of myocardial ischemia.

It was subsequently learned that in 1950 Grant [12] and associates had suggested splenic-artery substitution in treatment of hepatic artery aneurysms and that Glenn [11] had used the splenic artery to reestablish aortic continuity in a case of coarctation of the abdominal and lower thoracic aorta. In the latter instance, the splenic artery was used in continuity, the spleen resected, and the distal splenic artery anastomosed to the aorta proximal to the segment of narrowing.

In December, 1948, Olim [20] reported a case of coarctation of the aorta at the level of the diaphragm. A 17 mm. segment of stenotic aorta was resected; but even after extensive mobilization, the aortic ends could not be approximated, and the procedure was abandoned. Splenic-artery grafting in continuity might have been feasible in this case. It was also learned that Freeman [9] has anastomosed the splenic artery to the left common iliac artery prior to resection of an abdominal aortic aneurysm.[18] He also reported on substitution of the left superficial femoral artery for the right common

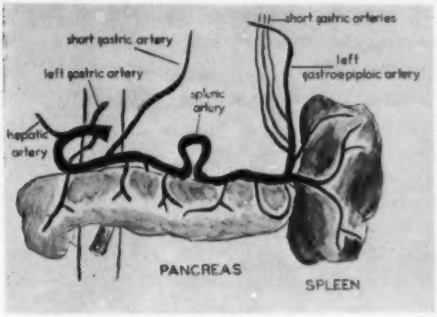


Figure 1

femoral artery in a case of thrombosed external iliac artery.[9]

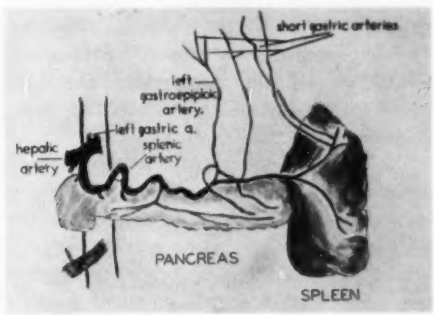
Thompson and Smithwick [24] have reported a case of a 34-year-old white man with hypertension. At second-stage left thoracolumbar splanchnicectomy, a nonpulsating left renal artery was found. The splenic artery was anastomosed to the distal renal artery. Seventeen days later, when it had been determined that renal function was nil, a left nephrectomy was performed. At this time, the blood pressure dropped significantly.

At the June, 1952, meeting of the Society for Vascular Surgery, Hurwitt [14] reported on fabrication of aortic grafts from split segments of splenic artery with good results.

Materials and Methods

Shumacker and Lowenberg,[22] and others [19] showed that the success of arterial end-to-end suture is contingent to a large extent upon the cross-sectional diameter of the vessels employed. Under fixed conditions, the likelihood of thrombosis or other complications following end-to-end suture rapidly mounts when vessels of 3.2 mm. outside diameter or less are used. Therefore, an outside diameter of 4 mm. was arbitrarily selected as the minimal size for reconstruction. Dissections were completed on 29 cadavers, 27 male and 2 female, commencing at the origin of the splenic artery from the celiac axis. Record was kept of main stem and branch lengths at which the vessel ceased to be 4 mm. in outside diameter. Three typical arterial arrangements are shown in Figures 1, 2, and 3, which were drawn from the dissections and reproduced diagrammatically.

Figure 2



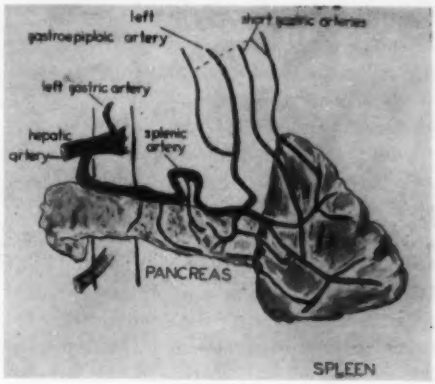


Figure 3

To assess the value of the splenic artery for reconstructive surgery, the artery was passed through an arc with its origin intact and its distal end detached. A cut in the diaphragm allowed us to determine that of 21 splenic arterial branches measuring at least 4 mm. in diameter, 17 reached to T8 or T9. In the caudad direction, the same number reached inferiorly to L4 or L5. Of 18 vessels 5.5 mm. in diameter, 15 reached the same level. In vessels 7-11 mm. in diameter, 50% were found to be just as long; the other half varied in shorter lengths. These data are summarized in Table 1 and Table 2.

The lengths of splenic artery of 4 mm. diameter or greater ranged from 6.7 to 27.5

TABLE 1.—*Frequency with Which Splenic Artery and Branches (Pulled Through the Diaphragm) Reach Specific Vertebral Levels in a Total of Seventy-Four Vessels* (Craniad Direction)*

	No.		No.		No.
T1-T2	1	T7-↓	2	T9-T10	8
T2-M	1	T7-T8	2	T10-↑	3
T2-T3	1	T8-↑	2	T10-M	3
T5-↑	1	T8-M	2	T10-↓	3
T5-↓	1	T8-↓	6	T10-T11	3
T5-T6	1	T8-T9	4	T11-↑	3
T6-M	1	T9-↑	5	T11-M	5
T6-T7	3	T9-M	6	T11-↓	2
T7-M	4	T9-↓	1		

* All arterial branches recorded had an outer diameter of 4 cm. or greater.

↑ = upper; ↓, lower, and M, middle portion of centrum.

cm., the average length being 15.3 cm. The two female cadavers had two of the longest splenic arteries found. Incidentally, in only one instance was the segment less than 10 cm. long. Frequently, a suitable cross-sectional diameter of splenic artery was found far into the hilus of the spleen or well beyond the origin of the left gastroepiploic artery (Table 3).

Specific studies were not done to determine the origin of all the arterial supply of the pancreas. Our findings, however, would seem to indicate that the anastomoses between pancreatic vessels of various origins are rich. Removal of the splenic arterial branches, therefore, would not seem to result in pancreatic ischemia.[1] The number of "anomalous" branches from the splenic artery was also of great interest, and should add to the rich collateral circulation of the pancreas. An "anomalous" origin of pancreatic arteries occurred with surprising frequency.

TABLE 2.—*Frequency with Which Splenic Artery and Branches Reach Specific Vertebral Levels in a Total of Seventy-Four Vessels* (Caudal Direction)*

	No.		No.		No.
S3-↓	1	L5-M	2	L3-↑	3
S2-↓	1	L5-↓	8	L3-M	6
S2-M	1	L4-L5	4	L3-↓	1
S1-↑	1	L4-↑	6	L2-L3	2
S1-M	3	L4-M	7	L2-↑	2
S1-↓	4	L4-↓	8	L2-M	1
L5-S	3	L3-L4	5	L2-↓	1
L5-↑	4				

* All arterial branches recorded had an outer diameter of 4 cm. or greater.

↑ = upper; ↓, lower, and M, middle, portion of centrum.

Comment

The splenic artery should indeed prove good material for arterial replacement. The diameter and length of average splenic arterial segments as found in these dissections should be adequate for arterial grafting. Lengths of this artery can be mobilized to any portion of the abdominal cavity, the groin, and the supradiaphragmatic regions as well. The tortuosity of this vessel, which has been described elsewhere,[6,18] carries the advantage of extra lengths of artery and lengthens the radius of operative intervention.

TABLE 3.—*Relation of Splenic-Artery Diameter to Number of Lateral and Terminal Branches*

Diameter, Mm.	No. of Branches
4	21
5	18
6	12
7	8
8	7
9	4
10	3
11	1

The technique of vascular suture best suited to any particular situation will necessarily vary. In general, an interrupted everting mattress suture gives the most consistently satisfactory results.[22] A continuous everting mattress suture proves almost as satisfactory for vessels of 5 mm. diameter and greater. Braided arterial silk 00000 or 0000 on straight or curved Atraumatic needles is uniformly acceptable. The finer points of technique are described elsewhere.[22]

The central location of the splenic arterial origin multiplies the possible uses to which this vessel may be put. It is long enough in desirable diameter to be used in surgery of the lower extremities, pelvis, and entire abdomen. Further, it may be taken through

a rent in the diaphragm and then used in procedures designed toward revascularization of the myocardium. The practical application of this in infradiaphragmatic surgery has already been shown by the work of Glenn, Freeman, and Thompson. It is hoped that the anatomical findings cited herein will encourage others to follow suit in using the splenic artery for infra- and supradiaphragmatic arterial grafting in continuity. Where radical cancer surgery necessitates arterial sacrifice, this artery may serve as a substitute.

The splenic artery may be used as a free arterial graft as well. In regions distant from the celiac axis, the vessel may be spliced into place after splenectomy. Uses to which it may be put to advantage include carotid, subclavian, brachial, iliac, femoral, and possibly popliteal arterial reconstruction. Replacement may be attempted in instances of trauma, either surgical or other, aneurysm formation, and progressive arterial obliteration.

Summary

1. The special anatomy of the splenic artery is described, with data regarding splenic artery lengths and calibers.

2. Some problems of arterial vascular grafting are described briefly, along with their solutions and their shortcomings.

3. A novel solution to this problem which utilizes the splenic artery as an autologous graft is given, as well as brief experience with this method. The splenic artery may be used in continuity or as a free arterial graft. It has potential for grafting peripheral, as well as visceral, arterial trunks, in aortic surgery, and for revascularizing the myocardium.

4. A challenge is extended in the hope that further attempts to use this method of arterial substitution will be made on clinical and laboratory levels.

245 Edwards St.

REFERENCES

1. Baronofsky, I. D.; Walton, W., and Noble, J. F.: Occult Injury to the Pancreas Following Splenectomy, Surgery 29:852-857, 1951.

2. Blakemore, A. H.; Lord, J. W., Jr., and Stefko, P. L.: The Severed Primary Artery in War Wounded: Non-Suture Method of Bridging Arterial Defects, Surgery 12:488-508, 1942.

3. Brown, R. B.; Hufnagel, C. A.; Pate, J. W., and Strong, W. R.: Freeze-Dried Arterial Homografts: Clinical Application, Surg. Gynec. & Obst. 97:657-664, 1953.

4. Carrel, A.: La Technique operatoire des anastomoses vasculaires et la transplantation des viscéres, Lyon méd. 98:859-864, 1902.

5. Cousar, J. E., III, and Lam, C. R.: Rectus Sheath Grafts in Vascular Repair, A. M. A. Arch. Surg. 65:471-876, 1952.

6. Cunningham, D. J.: Textbook of Anatomy, Ed. 7, edited by J. C. Brash and E. B. Jamieson, London, Oxford University Press, 1937, pp. 1207-1210.

7. Deterling, R.; Blunt, J. W.; Parshaley, M. S., and Hui, K.: A Critical Study of Present Criteria Governing Selection of Blood Vessel Grafts, read at meeting of Society of Vascular Surgery, Chicago, June 8, 1952.

8. Deterling, R. A., Jr.: Recent Advances in Vascular Surgery, Arch. Surg. 55:31-50, 1947.

9. Freeman, N.: Discussion, meeting of Society for Vascular Surgery, Chicago, June 8, 1952.

10. Gerber, A. B.; Lev, M., and Goldberg, S. L.: The Surgical Anatomy of the Splenic Vein, Am. J. Surg. 82:339-343, 1951.

11. Glenn, F.; Keefer, E. B. C.; Speer, D. S., and Dotter, C. T.: Coarctation of the Lower Thoracic and Abdominal Aorta Immediately Proximal to Coeliac Axis, Surg. Gynec. & Obst. 94:561-569, 1952.

12. Grant, J. L.; Fitts, W. T., Jr., and Ravdin, I. S.: Aneurysm of the Hepatic Artery—Report of 2 Cases and a Consideration of Surgical Treatment, Surg. Gynec. & Obst. 91:527-532, 1950.

13. Gross, R. E.; Bill, A. H., Jr., and Peirce, E. C., III.: Methods for Preservation and Transplantation of Arterial Grafts, Surg. Gynec. & Obst. 88:689-701, 1949.

14. Hurwitt, E., and Kantrowitz, A.: Construction and Use of Fresh Autogenous Arterial Transplants: II. Bridging of Aortic Defects by Splenic Artery Grafts, read at meeting of Society for Vascular Surgery, Chicago, June 8, 1952.

15. Johnson, J., and Kirby, C. K.: Vein Grafts in Coarctation of Aorta, read at meeting of Society for Vascular Surgery, Chicago, June 8, 1952.

16. Lowenberg, R. I.: Ureteral Segments Used for Homologous Arterial Grafting in Dogs, to be published.

17. Lowenberg, R. I., and Shumacker, H. B., Jr.: Experimental Studies in Vascular Repair: II. Strength of Arteries Repaired by End-to-End Suture, with Some Notes on Growth of Anastomosis in Young Animals, Arch. Surg. 59:74-83, 1949.

18. Michels, N. A.: The Variational Anatomy of the Spleen and the Splenic Artery, Am. J. Anat. 70:21-72, 1942.

19. Miller, H. H.; Callow, A. D.; Welch, C. S., and MacMahon, H. E.: The Fate of Arterial Grafts in Small Arteries: An Experimental Study, Surg. Gynec. & Obst. 92:581-588, 1951.

20. Olim, C. B.: Coarctation of the Aorta at the Level of the Diaphragm: Case Report, Ann. Surg. 130:1091-1097, 1949.

21. Payr, E.: Beiträge zur Technik der Blutgefass- und Nervennaht nebst Mittheilungen über die Verwendung eines resorbierbaren Metalles in den Chirurgie, Arch. klin. Chir. 62:67, 1900.

22. Shumacker, H. B., Jr., and Lowenberg, R. I.: Experimental Studies in Vascular Repair: Comparison of Reliability of Various Methods of End-to-End Arterial Sutures, Surgery 24:79-89, 1948.

23. Swan, H.; Robertson, H. T., and Johnson, M. E.: Arterial Homografts: The Fate of Preserved Aortic Grafts in Dogs, Surg. Gynec. & Obst. 90:568-579, 1950.

24. Thompson, J. E., and Smithwick, R., Jr.: Human Hypertension Due to Unilateral Renal Disease, with Special Reference to Renal Artery Lesions, Angiology 3:493-505, 1952.

25. Touroff, A., and Nabatoff, R.: Discussion, meeting of Society for Vascular Surgery, Chicago, June 8, 1952.

26. Tuffier, T., and Carrel, A.: Étude anatomopathologique et expérimentale sur le chirurgie des orifices due cœur, Presse méd. 1:173-177, 1914.

27. Vineberg, A. M.: Development of an Anastomosis Between the Coronary Vessels and a Transplanted Internal Mammary Artery, Canad. M. A. J. 55:117-119, 1946.

28. Weiss, E. W., and Lam, C. R.: Tantalum Tubes in the Non-Suture Method of Blood Vessel Anastomosis, Am. J. Surg. 80:452-454, 1950.

29. Williamson, C. S., and Mann, F. C.: Functional Survival of Autogenous and Homogenous Transplants of Blood Vessels, Arch. Surg. 54:529-538, 1947.

30. Woodburne, R. T., and Olsen, L. L.: The Arteries of the Pancreas, Anat. Rec. 111:255-270, 1951.

Editorial

Postoperative Wound Infections

Judging from current literature and discussions at medical meetings, postoperative wound infections are becoming of sufficient frequency and seriousness to justify everyone's attention. Altemeier in his recent report to the Governors of the College of Surgeons covered the subject very completely. He decried the widespread use of antibiotics for conditions for which they have been proved to be of no specific value. The common cold, I believe, is one of the best examples. Prophylactic use of antibiotics in clean surgical cases should be discontinued.

All hospitals should have a committee to investigate means of prevention as well as to investigate wound infections when they occur. It is better to prevent such infections by rigid observation of recognized methods than it is to try and eradicate such antibiotic-resistant bacteria from hospitals once they have become firmly entrenched.

It seems generally agreed that the increased incidence of infection has resulted from increasing resistance of bacteria to antibiotics, staphylococci in particular, and in a general letdown in aseptic and antiseptic techniques. It is the latter problem which I wish to discuss briefly.

Some observers believe that these organisms have not increased in virulence. If virulence has not increased and the incidence of postoperative infections have increased over that of the preantibiotic era, one can only conclude that our aseptic surgical techniques have undergone deterioration. That this has occurred has been evident to me for several years. Observations in surgical clinics over the various parts of the mainland United States and in other countries, as well as contact with the house staffs of our local hospitals, recruited from widely dispersed medical centers, lead me to believe that many of the principles laid down by Lister and others, regarding asepsis and antisepsis, are being sadly neglected.

Entirely too much reliance is being placed on the protection expected from antibiotics and the direct action of other antibacterial agents. Frequent inquiry of recent graduates as to their knowledge of Lane technique, which was widely used in operations on bones and joints (so susceptible to infection, in earlier years), reveals complete ignorance of this subject. I am sure only we of older vintage have ever witnessed an operation done in which no part of any instrument or other foreign body introduced into a wound had been touched by any object after sterilization. Such rigid technique may not be necessary today, but it certainly instilled into the operating team a surgical reflex one now sees so frequently neglected.

Some of the points of surgical technique that seem important to me, and which are frequently observed to be violated by nurses and physicians alike are as follows:

1. A three-minute or less handscrub using some preparation of hexachlorophene of varying strength is accepted by many as being sufficient. While it is true that some persons can accomplish more in one minute with a brush than

others do in many minutes, I still believe mechanical cleansing of hands by initial scrub of ten minutes should be insisted upon. One should always remember that the donning of sterile gloves is no excuse for inadequate hand preparation. Too many holes occur in gloves during the course of an operation to justify such lack of care.

2. While there is no standard method of preparing an operative field, mechanical cleansing of the area seems the most important to me, and the type of antiseptic used is probably of only secondary importance. More attention to this phase of the operation needs emphasis.

3. It was the teaching some years ago that skin harbored bacteria and that it should be excluded from the operative field. In more recent years, this teaching has been largely disregarded. Large areas of skin are frequently left uncovered. Viscera are spread out over the skin with abandon. Sutures are dragged across this area, and the gloves of the surgeon and those of his assistants meet skin under the friendliest of terms. Some surgeons, after the operation is well under way, cover the skin in a desultory sort of fashion—too late and too incompletely to be of much value. I have been told that once skin drapes become wet, they are more detrimental than useful. So far I do not know of any convincing proof to show that such is the case. If it is, there are methods of protecting the skin by drapes impervious to moisture.

4. Even though the intestinal contents can be largely sterilized with adequate administration of antibiotics preoperatively, one should be careful to prevent soiling of the operative field from this source. Soiled instruments, abdominal pads, gloves, and other contaminated material should be discarded promptly. Many nurses seem inadequately taught how to keep contaminated instruments quarantined from the main source of supply. It is well to remember that a wound may be able to resist a small amount of exposure to contaminated material, but unable to resist larger amounts.

5. Speed in performing surgical operations was once thought to be the mark of a good surgeon. With good anesthesia and other supportive measures by which the patient can be surrounded, speed has assumed much less importance. There has been a tendency in some quarters, I believe, to err in the opposite direction. No one should ever sacrifice safety for speed, and neither should an operation be prolonged unnecessarily by dillydallying. Trauma to tissues and overlong exposure to sources of contamination have long been recognized as fruitful avenues of infection. With the high cost of operating room care, the economic factor, if no other, should be considered.

6. Another source of wound infection, which I believe is of great importance, is the handling of infected material by doctors and nurses alike with the unprotected hand. Certainly it is better to keep one's hands free of infectious organisms than it is to try to eliminate them by scrubbing and application of antiseptics. It has been pointed out by some that the infection rate on wards where the personnel is usually less well trained is greater than it is on floors where private patients are cared for by the more experienced.

7. The importance of screening personnel with infections from the operating room seems less well recognized than formerly. Persons with furuncles and throat infections, and carriers of pathogenic germs that may be identified by proper cultural methods, are still sources to be reckoned with even in this antibiotic era.

There are many sources of wound infection that have not been touched upon, but the ones mentioned seem the ones, from my observations, most flagrantly neglected. One wonders at times if aseptic surgical techniques, like the bustle and the horse and buggy doctor, have gone out of fashion.

J. E. Strode, M.D.
Straub Clinic
Honolulu

Case Reports

Triplication of Gallbladder

WELLAND A. HAUSE, M.D., Decatur, Ill.

Reduplication of the gallbladder and cystic duct are rare anatomic anomalies. Recently Skielboe [1] has reviewed the literature and reported a case of triple gallbladder, the first such recorded. Because of the rarity of the condition, it is felt worth while briefly to report another case.

Report of Case

A 69-year-old woman had her first attack of gallbladder pain 11 months before surgery. There were several other attacks during the period; they were accompanied by nausea and vomiting, but no jaundice was observed. X-ray study of the gallbladder revealed poor concentration of the dye and numerous calculi. At surgery, the gallbladder was removed without particular difficulty, although a few filmy adhesions surrounded it. The fact that is was a triplex organ was not recognized until it was examined in the laboratory.

Dissection revealed an organ which measured 9 cm. in length and up to 6 cm. in diameter. There were three separate chambers, the two larger being each approximately the size of the usual gallbladder and sharing a common middle wall. Each contained thin dark brownish-green bile and had a separate cystic duct, which extended to the line of excision. The third chamber lay superior to the first two and was not covered with serosal membrane. It was considerably smaller and contained mucoid bile and 20 dark-brown, smooth-surfaced calculi, varying from 0.2 to 2.5 cm. in diameter. The mucosa here was coarsened and irregular and the wall thick and tough. Its cystic duct could be opened only partially with the scissors. Microscopically, the chamber containing the calculi showed moderate chronic inflammation of the wall; the others presented about the usual gallbladder architecture. The usual layers of mucosa, muscularis, and subserosa were seen in the walls of all three chambers.

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Pathologist, St. Mary's Hospital.

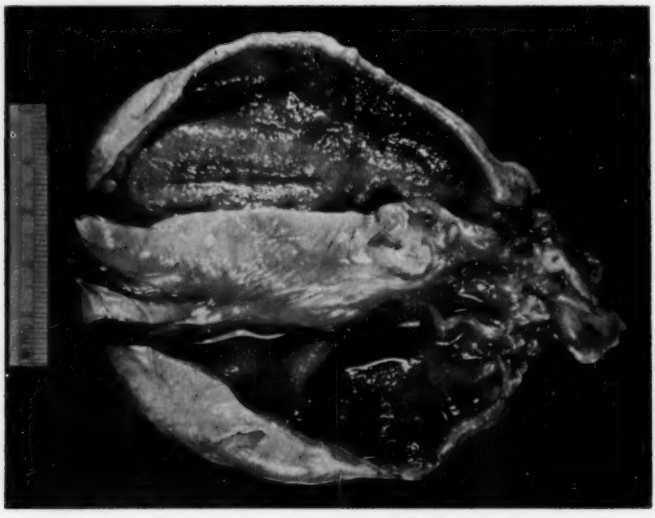

Fig. 1.—Posterior-inferior surface of gallbladder, showing two grossly normal chambers.

Fig. 2. — Gallbladder with three opened chambers. Note thickened wall of one containing calculi.

The accompanying photographs demonstrate the nature of the specimen.

A case of triplication of the gallbladder is reported.

REFERENCE

1. Skielboe, B.: Anomalies of the Gallbladder—Vesica Fellea Triplex: Report of a Case, Am. J. Clin. Path. 30:252-255 (Sept.) 1958.

Sigmoid Colon Pull-Through Procedure for Rectovaginal Fistula

W. W. GLAS, M.D., Eloise, Mich.

Rectovaginal fistula following a combined radiological-surgical attack upon carcinoma of the endometrium or following radium therapy of the cervix is a complication of infrequent occurrence,[1] but of considerable significance. This complication is particularly unfortunate when the patient is cured of the carcinoma. It is generally agreed that rectovaginal fistulas are rare following treatment for carcinoma of the corpus. A technical error in irradiation or a posthysterectomy pelvic abscess is the most frequent cause of fistula following the treatment for carcinoma of the corpus. Rectovaginal fistula is a more frequent complication following treatment for carcinoma of the cervix, due to neoplastic invasion of the rectovaginal septum.

Many procedures have been described for the surgical treatment of this complication.[2,3] All of these procedures depend upon mobilization of intensely irradiated tissues which are fibrotic and extremely hard to dissect, in addition to which there is poor healing. Recurrences following surgical intervention for rectovaginal fistula are frequent.

The purpose of this paper is to report a new technique for the repair of rectovaginal fistula following irradiation and hysterectomy for carcinoma of the endometrium. This same technique has been used recently in a second patient with rectovaginal fistula following x-ray therapy for carcinoma of the cervix.

Report of Case

A 41-year-old white woman was admitted to Wayne County General Hospital on June 4, 1957, with a single-barreled sigmoid colostomy and a rectovaginal fistula. In 1952 a diagnosis of carcinoma of the endometrium of the uterus was established, after which she was given x-ray and radium therapy. Five weeks later an abdominal hysterectomy was carried out. She returned seven days after her discharge complaining of rectal pain. Examination revealed evidence of a cul-de-sac abscess. One month later a rectovaginal fistula developed spontaneously. This was treated by a single-barreled sigmoid colostomy on Jan. 5, 1953. The patient had an uneventful recovery and was seen intermittently during the next four years. There was no evidence of recurrence of the carcinoma. She was extremely concerned about the colostomy and desired repair of the fistula to permit gainful employment. Examination on June 4, 1957, revealed a defunctionalized rectum. There was a rectovaginal fistula, 1.5 cm. in diameter, located at the proximal end of the posterior vagina. The mucosa of the vagina was atrophic. The anal sphincter was normal, with good tone. There was a stenosis of the rectum at the level of the fistula. A barium study of the colon proximal to the colostomy was normal.

On June 28, 1957, a resection of the blind stump of the defunctionalized rectosigmoid was carried out. Because of the perirectal thickening and fibrosis due to irradiation, it was impossible to dissect the tissues at the level of the fistula. The rectum was amputated at the level of the fistula (Figure), and the remaining rectal mucosa was curetted distally as far as possible. The proximal rectum, rectosigmoid, and sigmoid colon distal to the colostomy were then mobilized and resected. To facilitate this, the inferior mesenteric artery was ligated and transected at its origin from the aorta. The colostomy stoma was mobilized and excised and the descending colon mobilized, preserving the left colic and the marginal artery. With the patient in the lithotomy position, the descending colon was pulled through the rectal ampulla and anus, with 10 cm. of redundant colon protruding beyond the anus (inset *A*, Figure). Particular care was taken not to compromise the vascularity of the mobilized colon. The anal sphincters were gently dilated prior to the pull-through procedure. Closure of the abdominal wound was carried out in the usual manner. The patient experienced an uneventful postoperative course and on the fourth

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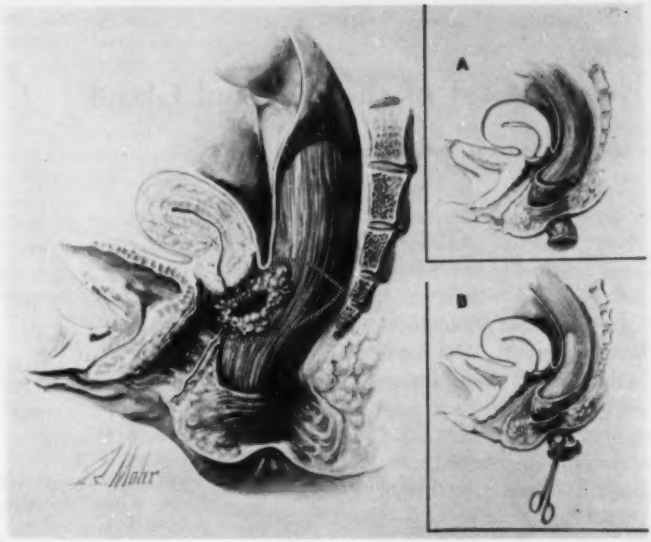
From the Department of Surgery, Wayne County General Hospital.



Rectovaginal fistula following irradiation.

(*A*) The mobilized descending colon is shown protruding through the anus. The rectum has been amputated at the level of the fistula.

(*B*) The redundant colon is amputated just inside the anus 14 days following the above procedure.

In these illustrations the uterus has been drawn in by the artist. In the case report (see text) the uterus had been removed.

postoperative day passed gas and feces through the perineal colostomy. On the 14th postoperative day the redundant descending colon was amputated 1 cm. inside the anus (inset *B*, Figure). Upon the patient's discharge 10 days later, the colostomy stoma had retracted approximately 1 cm. and could be palpated easily in the rectal ampulla. The patient was instructed to carry out rectal examination and stomal dilatation once monthly.

Check-up visits during the past 12 months have revealed the patient to have excellent bowel control. There is no evidence of recurrence of the fistula or the carcinoma. She is at the present time gainfully employed.

Examination at the present time reveals a slight pouching of the serosa of the colon at the site of the rectovaginal fistula. This has not caused the patient any difficulties. The colostomy stoma is easily palpable upon rectal examination and has remained patent.

This form of treatment of the rectovaginal fistula is based upon sound surgical principles and is similar to the abdominoendorectal pull-through operation described by Black and Botham.[4] Irradiated tissues show slow or poor healing. This, combined with extreme inelasticity due to fibrosis, prevents adequate mobilization of the tissues necessary for repair. With the mobilization of the descending colon, an entire new gastrointestinal tract can be rotated into position so as to replace the diseased proximal rectum and rectosigmoid colon. No sutures were necessary to hold the mobilized descending colon in the pelvis, since the operative manipulation itself caused the colon to become fixed in position. Although there was a small segment of overlapping bowel in the rectal ampulla, this has not caused any difficulties.

Summary

A new technique for repair of a rectovaginal fistula following hysterectomy and irradiation, with the report of a case, is presented. This consisted of mobilization of the left colon and amputation of the rectum as low as possible, with preservation of the anus. The mobilized colon was then pulled through the anus. Fourteen days later the redundant colon was amputated just inside the anus. A two-year follow-up has revealed a satisfactory result.

Wayne County General Hospital.

REFERENCES

1. Lampe, I.: Personal communication to the author, November, 1958.

2. Te Linde, R. W.: Operative Gynecology, Ed. 2, Philadelphia, J. B. Lippincott Company, 1953, pp. 254-262.

3. Greenhill, J. B.: Handbook of Surgical Gynecology, Chicago, Yearbook Publishers, Inc., 1953, p. 200.

4. Black, B. M., and Botham, R. J.: Combined Abdominoendorectal Resection: A Critical Reappraisal Based on 91 Cases, S. Clin. North America, Aug. 1957, pp. 989-997.

Benign Cyst of the Adrenal Gland

HARRY V. ARMITAGE, M.D., Chester, Pa.

Cysts of the adrenal gland occur with relative rarity. The first reported case has been credited to Greiselius,[1] of Vienna, who described the lesion in 1670. Henschen [2] stated that by 1906 there had been seven cases reported. Writing in 1947, Reimann and Guyton [3] were able to find only four papers during a 10-year period in the American literature on this subject. Probstein and Blumenthal,[4] in 1956, stated that there had been a total of 83 cases reported.

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Because of the rarity of this condition, it is believed worth while to report another case and to comment on some of the pertinent facts regarding the origin, diagnosis, and treatment of adrenal cysts.

Report of a Case

A 40-year-old white woman was admitted to Chester Hospital because of left renal colic and hematuria. An intravenous pyelogram was made and disclosed two or three small areas of calcification, which were thought to represent stones, in the right kidney. There was similar calcification in

Intravenous pyelogram showing mass above right kidney. The stones mentioned are not visualized in this reproduction.

the left kidney and a 3 mm. stone in the left ureter. In addition to these findings, there was a soft-tissue density, measuring approximately 25 to 30 cm. in diameter, lying above the level of the right kidney (Figure). After the pyelogram was made, the stone in the left ureter descended to the ureterovesical junction and was subsequently extracted through a cystoscope by Dr. Louis Iozzi.

It was then attempted to determine the nature of the soft-tissue mass above the kidney. A cholecystogram was made, and the gallbladder, which was normal, could distinctly be delineated from the mass. Special anteroposterior and lateral roentgenograms of the abdomen were made, and it was believed that the density lay posterior to the liver and above the right kidney. Its position could not be definitely ascertained, however, and the possibility of the mass being intra-abdominal was considered. Carbon dioxide was injected into the perirenal area in an attempt to outline the mass. Roentgenograms made at this time were unsatisfactory, and the position and nature of the mass were not definitely established.

It was believed that further study would be of little value and that surgical teratment was indicated. Accordingly, the abdomen was explored through a transverse incision, starting 1 in. above the umbilicus and extending to the right flank. The tumor was readily located in a retroperitoneal plane behind the liver and above the right kidney. The posterior peritoneum was incised, and the kidney was depressed. A cyst, which measured approximately 30 cm. in diameter, was removed with little difficulty. The cyst was lying in juxtaposition to the adrenal gland, but the gland itself was intact. The wall of the cyst was diffusely studded with yellow-orange tissue, which grossly resembled adrenal tissue. The lumen contained thin fluid, which was brown-green in color. Microscopic examination disclosed a non-neoplastic cyst with adrenal tissue in the wall. Further study at the Armed Forces Institute of Pathology confirmed the opinion that the lesion was a benign adrenal cyst.

The patient made an uneventful recovery from the operation and was discharged from the hospital eight days later.

Comment

The pathogenesis of adrenal cysts is difficult to trace. Trauma at birth or during later life has been implicated as a causative agent. Doran [5] states that a history of trauma may be elicited in more than one case. Trauma as a cause is particularly applicable to infants. The adrenal gland at birth is large and extremely vascular, and the inner cortex presents congestion, with fatty and some necrotic changes. Minimal trauma at this time may result in an adrenal hematoma, as reported by Marin et al.[6] Encapsulation of the hematoma could lead to the formation of an adrenal cyst, or, more properly, a pseudocyst.

Another category of adrenal cysts is said to be the result of ectasia of angiomatous cavities. Most of the large multiloculated cysts that have been reported have resulted from dilatation of lymph channels. Blood spaces may also be a site of origin. Probstein and Blumenthal [4] believe that many cysts originate as venous aneurysms in a process of maldevelopment. They believe that such cysts are particularly prone to intracystic hemorrhage.

Fiala and Rank [7] speculated that an adrenal cyst accompanied by hemorrhage had its genesis during pregnancy and was aggravated by another pregnancy. There seemed to be little basis for the first assumption.

Cystic degeneration of a cortical adenoma has been suggested as a possible mechanism of formation, but there is no real evidence to support this occurrence. However, Ellis and associates [8] described three cysts which had occurred as a result of degeneration of pheochromocytomas.

Echinococcus cyst of the adrenal is within the realm of possibility. Barnett [9] has been cited as finding adrenal involvement in less than 0.5% of cases of infestation. Actually, Barnett, after a study of 1,617 records of Echinococcus disease, failed to mention adrenal specifically but stated that a group classified as "other tissues" was involved in less than 0.5%.

It is not surprising that the majority of adrenal cysts have been found post mortem because, as a rule, they produce no symptoms unless they grow large enough to compress or displace surrounding organs. There have been, however, a number of symptoms attributed to adrenal cysts. Pearse [10] reported a case in which severe pain and shock occurred as a result of bleeding into the cyst. Probstein and Blumenthal [4] reported an instance of extensive

intracystic hemorrhage in which the erythrocyte count dropped from 4,500,000 to 2,000,000 within 24 hours. Flatulence and low-grade epigastric pain occurred in a case reported by Lloyd and Solomon.[11] These symptoms were attributed to displacement of the stomach by the cyst and were relieved when the cyst was removed. Ballance [12] reported a case in which indigestion and back pain were the presenting symptoms. Teasley et al.[13] ascribed pain in the lower part of the thorax to an adrenal cyst. Segal and Specter [14] reported a large palpable tumor in the left upper part of the abdomen, and others have discovered the lesion when it has grown large enough to be felt. Brindley and Chisholm [15] reported two cases of benign cystic tumors associated with Cushing's syndrome. At the other extreme, Moore and Cermak [16] reported adrenal cysts associated with adrenal insufficiency in an infant with fatal termination at the age of 7 months.

Adrenal cysts may vary from those minute in size to cysts the size of one reported by Gardiner et al.,[17] which had a total weight of 46 lb. (20.9 kg.) and contained 11 liters of fluid. Lymphangiectatic cysts are frequently loculated and contain clear or milky fluid. Hematocysts contain fluid which is yellow to brown.

The interior may be smooth or may contain irregular calcified areas, and the exterior may be marked by yellow bits of tissue, which on histologic study are found to be adrenal tissue. Unilateral cysts are twice as common as bilateral cysts, and women are affected twice as often as men (Rabson [18]).

The majority of cysts that are discovered during life are found fortuitously. Roentgenograms on occasion may lead to their discovery. Levison [19] demonstrated bilateral adrenal cysts by roentgenogram in 1933 and reported that this was the first time this method had been used for diagnosis. Teasley et al.[13] discovered an adrenal cyst because of calcification visible on a roentgenogram and further demonstrated the lesion by pneumograms. Flattening of the upper pole of the kidney, as seen in the urogram, may be an important diagnostic sign (present in the case being reported). Large cysts may displace the colon, stomach, spleen, or kidney. A large lymph cyst reported by von Gusnar [20] displaced the right kidney to the left side until it lay beside the left kidney.

While adrenal cysts have been treated by packing and draining the cavity, the ideal treatment is complete removal. Either a lumbar or an abdominal approach may be used. If it is definitely established that the mass is retroperitoneal, a lumbar approach would seem preferable. However, if there is a question concerning the site of origin of the tumor, an abdominal approach is indicated. On occasion a combined intra- and extraperitoneal approach has been used.

Summary

A case is presented in which a benign cyst of the right adrenal gland was discovered by roentgenogram and removed surgically. Comment is made on some of the criteria which might be helpful in the recognition and treatment of adrenal cysts.

400 E. 13th St.

REFERENCES

1. Greiselius, cited by Doran.

2. Henschen, K.: Über Struma suprarenalis cystica hæmorrhagica, Beitr. klin. Chir. 49:217-265, 1906.

3. Reimann, D. L., and Guyton, W. L.: Cysts of the Adrenal Gland with a Case Report, Am. J. Path. 23:479-483, 1947.

4. Probstein, J. G., and Blumenthal, H. T.: Adrenal Cyst with Extensive Intracystic Hemorrhage: Observations on Criteria for Clinical and Pathological Diagnosis, A. M. A. Arch. Surg. 73:1026-1030, 1956.

5. Doran, A. H. G.: Cystic Tumour of the Suprarenal Body Successfully Removed by Operation, with Notes on Cases Previously Published, Proc. Roy. Soc. Med. 1:201-220, 1907-1908.

6. Marin, H. M.; Graham, J. H., and Kickham, C. J. E.: Adrenal Hematoma Simulating Tumor in a Newborn: Report of a Case and Review of the Literature, A. M. A. Arch. Surg. 71:941-945, 1955.

7. Fiala, J. C., and Rank, R. K.: Hemorrhagic Cyst of the Adrenal Gland: A Case Report, South Dakota J. Med. & Pharm. 8:115-117, 1955.

8. Ellis, F. H., Jr.; Dawe, C. J., and Clagett, O. T.: Cysts of the Adrenal Glands, Ann. Surg. 136:217-227, 1952.

9. Barnett, L.: Hydatid Cysts: Their Locations in the Various Organs and Tissues of the Body, Australian & New Zealand J. Surg. 12:240-248, 1943.

10. Pearse, H. E.: Cyst of the Adrenals: Report of a Case, Tr. West. S. A. 26:329-336, 1916.

11. Lloyd, O. D., and Solomon, L.: Solitary Cyst of the Adrenal, Calif. Med. 74:437-439, 1951.

12. Ballance, H. A.: Cyst of the Right Suprarenal Capsule Removed by Operation, Brit. M. J. 1:926-928, 1923.

13. Teasley, G. H.; Good, L. P., and Klein, C. P.: Hemorrhagic Cyst, with Calcification, of the Adrenal Gland, M. Radiog. & Photog. 33:80, 1957.

14. Segal, M., and Specter, J.: Benign Cysts of the Adrenal: Report of an Unusual Case, J. Albert Einstein M. Center 5:42-45, 1957.

15. Brindley, G. V., Jr., and Chisholm, J. T.: Cystic Tumors of the Adrenals Associated with Cushing's Syndrome, Texas J. Med. 47:234-237, 1951.

16. Moore, F. P., II, and Cermak, E. G.: Adrenal Cysts and Adrenal Insufficiency in an Infant with Fatal Termination, J. Pediat. 36:91-95, 1950.

17. Gardiner, W. R.; Bell, H. G., and Althausen, T. L.: Large Adrenal Cystic Tumor Without Endocrine Manifestations, Postgrad. Med. 11:297-300, 1952.

18. Rabson, S. M., and Zimmerman, E. F.: Cystic Lymphangiectasia of the Adrenal, Arch. Path. 26: 869-872, 1938.

19. Levison, P.: A Case of Bilateral Adrenal Cysts, Endocrinology 17:372-376, 1933.

20. von Gusnar, K.: Nebennieren-Lymphcyste, Chirurg 21:497-499, 1950.

Metastatic Chordoma

Use of Mechlorethamine (Nitrogen Mustard) in Chordoma Therapy

AUSTIN J. McSWEENEY, M.D., and P. RICHARD SHOLL, M.D., Janesville, Wis.

The sacrococcygeal chordoma is a rare tumor which is generally believed to arise from remnants of embryonic notochordal tissues. These may persist as the nucleus pulposus or in scattered nodular rests in some 2% of humans.[1] A recent review of the literature revealed a total of 505 cases of chordoma.[2] Chordomas show a predilection for the sacrum and coccyx, and about half are located in these areas. Although chordomas are malignant tumors in every sense and are locally invasive, proved metastases are very uncommon. Apparently, metastases from mucus-producing carcinomas of the rectum have in some cases been confused with metastatic chordoma. We are aware of only one case of presumable distant bony metastasis of a chordoma, but this was not proved by histologic examination.[3]

The primary therapeutic approach to sacrococcygeal chordomas is surgical. Block removal of the coccyx, most of the sacrum, and the tumor is recommended if one desires to effect a permanent cure.[4] Nevertheless, complete extirpation of the malignant tissue is difficult, and recurrence is usual. X-ray therapy is of considerable value in the management of some chordomas, whereas others are little affected by irradiation. No other form of curative or palliative treatment for this malignant tumor is generally advocated.

In 1952 Snodgrass and Suckle reported a case of chordoma which they treated surgically.[5] Subsequent developments in this case warrant an additional report. The patient has manifested a consistently favorable response to repeated mechlorethamine (nitrogen mustard) therapy. Distant bony metastasis has occurred, as proved by histologic examination.

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From the S-P-N Foundation of the Pember Nuzum Clinic, 500 W. Milwaukee St.

Report of Case

A 78-year-old Caucasian woman has been observed in the Clinic since August, 1950, because of a sacrococcygeal chordoma. Roentgenologic examination originally showed an indefinitely outlined soft-tissue mass overlying the lower portion of the sacrum and the coccyx, and a destructive process involved these bones (Fig. 1). Surgical excision of a 250 gm. translucent, jelly-like tumor was carried out on Sept. 8, 1950, and microscopic sections revealed large physaliphorous cells in a matrix which stained positively for mucin (Fig. 2).

Postoperatively the patient did very well, and she was entirely comfortable until October, 1951, when reappearance of the tumor mass was first detected. Local excision of the recurrent chordoma was performed in June, 1952, and August, 1954, in vain attempts to relieve the patient's pain. In September, 1954, temporary comfort was achieved by local x-ray therapy. A year later radiation treatment was carried out to the limit of the patient's tolerance, with little benefit. In October, 1955, laminectomies were performed at levels L-3 through L-5, and the recurrent chordoma was again resected. Pain returned quickly; and in June, 1956, the recurrent chordoma and adjacent sacral tissue were excised surgically. In October and November, 1956, and in March, 1957, chordotomies were performed, in a desperate attempt to relieve the patient's intractable and severe pain. Notwithstanding these heroic efforts, her life was one of almost continuous anguish, due to pain over the sacrum and in the lower extremities. Morphine injections at four-hour intervals afforded her only partial relief from pain. In spite of the torment to which this pitiable patient had so long been subjected, her general physical condition remained excellent.

A trial of intravenous mechlorethamine therapy was first initiated on Dec. 14, 1957. Our patient, who weighed 54.4 kg., was given a total of 25 mg.

Fig. 1.—Roentgenogram of pelvis, showing primary site of sacrococcygeal chordoma.

of mechlorethamine hydrochloride, divided into three daily doses. She first experienced diminution of pain on Dec. 19; and she eventually became totally free of pain. It was possible to eliminate therapy with morphine gradually, although the patient had become addicted to this drug. Subsequent recurrences of pain in the sacrum and lower extremities have made it necessary to repeat treatment with mechlorethamine at intervals of four to seven weeks. Her most recent course commenced

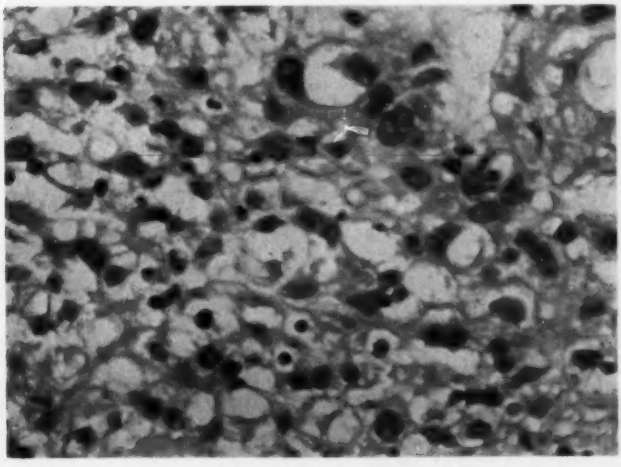

Fig. 2.—Section of sacrococcygeal tumor after resection. Note the large, clear, closely packed cells, many of which are vacuolated. × 400.

Fig. 3.—Metastatic chordoma of left ulna. While sacrococcygeal chordomas occasionally spread to the adjacent lumbar spine, no other instance of proved distant bony metastasis is known.

on Sept. 22, 1958; and in every case the patient has had very gratifying relief of her pain. No improvement has been noted in the x-ray appearance of the tumor-infiltrated sacrum and lumbar spine since she has been receiving mechlorethamine therapy.

In August, 1958, the patient first noted pain and tenderness in the left forearm, which she ascribed to a fall; however, a tender tumor mass soon became apparent at the junction of the middle and the distal third of the shaft of the ulna. Roentgenologic examination of the left forearm on Aug. 28 showed a neoplasm in the shaft of the ulna, approximately 6 cm. from the distal end. There was cortical thinning and elevation of the periosteum with secondary new-bone formation evident outside the cortex. Fine trabeculations were present in the new bone. Within the tumor there was a central translucent area, measuring approximately 1 cm. in its widest dimension (Fig. 3).

On Sept. 3, 1958, the bone tumor was excised and a Steinmann pin was inserted for stabilization of the ulna. Histologic examination of the tumor showed it to be similar to that removed from the sacrococcygeal area originally in 1950. The neoplasm was made up of small, irregular masses and sheets of round, oval, and polyhedral cells, separated by homogeneous pale-blue-staining ground substance and supported in places by a small amount of fibrous connective-tissue stroma. Many of the tumor cells were vacuolated (Fig. 4).

Summary

Eight years of therapy in a case of sacrococcygeal chordoma have been reviewed. This case of chordoma is unique in that distant bony metastasis has been proved by histologic study. Periodic mechlorethamine (nitrogen mustard) therapy has afforded the patient dramatic and gratifying relief of

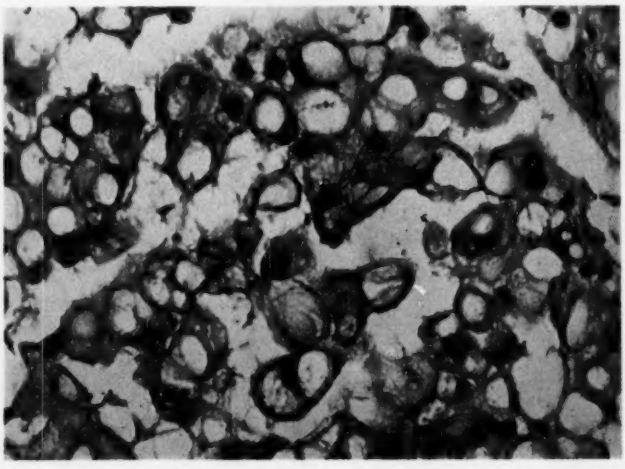

Fig. 4. — Photomicrograph of metastatic chordoma after excision from left ulna. The histologic features of this tissue are almost identical with those of the primary tumor. Physaliphorous cells are abundant and easily identified. × 400.

pain in the sacrum and lower extremities. Although mechlorethamine did not have curative value in our case, its further trial in the management of pain in recurrent chordoma is in order.

Pember Nuzum Clinic, 500 W. Milwaukee St.

REFERENCES

1. Shepherd, J. A.: Sacrococcygeal Chordoma, Brit. J. Surg. 175:516-519 (March) 1955.

2. Utne, J. R., and Pugh, D. G.: The Roentgenologic Aspects of Chordoma, Am. J. Roentgenol. 74:593-608 (Oct.) 1955.

3. MacCarty, C. S.: Personal communication to the authors.

4. Dahlin, D. C., and MacCarty, C. S.: Chordoma: Study of 59 Cases, Cancer 5:1170-1178 (Nov.) 1952.

5. Snodgrass, T. J., and Suckle, H. M.: Sacrococcygeal Chordoma, A. M. A. Arch. Surg. 65: 181-187 (July) 1952.

Kartagener's Syndrome

LIEUT. COL. BILLIE G. STREETE (MC) and MAJOR FRED E. STULL JR. (MC), U. S. A. F.

The purpose of this paper is to report a case of Kartagener's syndrome. The occurrence of situs inversus and bronchiectasis was first noted in 1904 by Siewert,[4] who described a case of situs inversus in a 20-year-old youth. The triad of situs inversus, bronchiectasis, and paranasal sinusitis has been more fully described by Adams and Churchill.[1] Kartagener[2] reported 11 cases in 1933 and 1935.

The reason for the occurrence of bronchiectasis in patients who have situs inversus has never been explained, but the associated higher incidence has been clearly shown.[3]

Report of Case

The patient is a 14-year-old white girl who entered the hospital on July 7, 1958, with a history of productive cough "all her life." The sputum was described as yellow and not foul. The patient's mother had had no viral disease during her pregnancy. The patient has one brother and one sister, and there is no family history of situs inversus. The malposition of the heart was discovered when the child was one year of age. She had had repeated upper respiratory infections, as well as pneumonia three times. Frontal headaches led to the diagnosis of pansinusitis. There was no history of weight loss, hemoptysis, or night sweats. Allergy work-up revealed the patient to be allergic to feathers.

Because of the chronic productive cough, bilateral bronchograms were made and revealed severe cystic bronchiectasis of a contracted left middle lobe and mild cylindrical bronchiectasis of the right basal segments.

Physical examination revealed a well-developed girl, who spoke in a nasal voice. Blood pressure was 105/60; pulse 70 per minute, and temperature 98.6 F.

The apex of the heart was found to be in the midclavicular line on the right side of the chest. Liver dullness was noted on the left side to percussion.

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Fig. 1.—Severe bronchiectasis of the "left middle lobe."

Laboratory Studies.—A white blood cell count was 13,400, with 67% neutrophils, 32% lymphocytes, and 1% eosinophils. Hemoglobin was 12.5 gm. per 100 ml. Sedimentation rate was 14 mm. in one hour. Sputum cultures were negative for pathogenic organisms. Urine was normal. Roentgenograms of the chest revealed dextrocardia and infiltration of the left middle lobe area and the

Fig. 2.—Bronchiectasis of right basal segment.

Fig. 3.—Dextrocardia with bilateral pulmonary infiltrate.

right base. The stomach bubble was on the right side. The electrocardiogram was typical of dextrocardia. Sinus x-rays revealed diffuse paranasal pansinusitis. With the rather localized severe bronchiectasis of the left middle lobe, it was felt

Fig. 4.—Electrocardiogram showing dextrocardia.

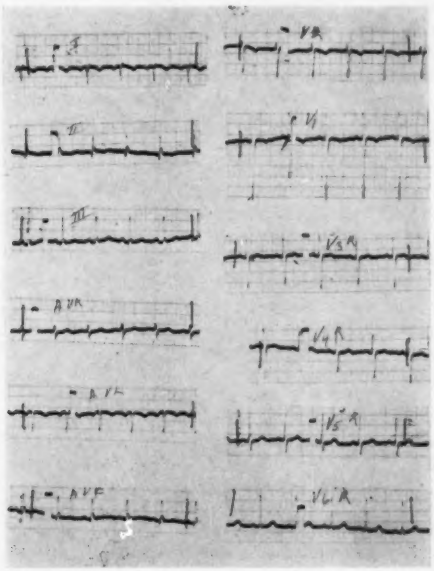

Fig. 5.—Left maxillary sinusitis.

that this patient would benefit from surgery. On July 11, a left thoracotomy was performed. The left lung consisted of three lobes. The upper and lower lobes appeared to be normal. The middle lobe was rather small and diseased. The anatomy here was like that usually encountered in the right chest. The left middle lobe was removed without difficulty. The patient's postoperative course was benign, and follow-up after six months revealed marked improvement in her symptoms.

Summary

Over 100 cases of Kartagener's syndrome have been described. This case adds to the smaller number who have been treated surgically with improvement.

U. S. A. F. Hospital, Wiesbaden, Germany, APO 633, New York (Lieutenant Colonel Streete).

REFERENCES

1. Adams, R., and Churchill, E. D.: Situs Inversus, Sinusitis, Bronchiectasis: Report of 5 Cases, Including Frequency Statistics, J. Thoracic Surg. 34:206-217 (Dec.) 1937.

2. Kartagener, M., and Ulrich, K.: Zur Pathogenase der Bronchiektasien; Bronchiektasien und Veränderungen der Nasennebenhöhlen, Beiträge klin. Tuberk. 86:349-357, 1935.

3. Richards, W. F.: Situs Inversus Viscerum, Absent Frontal Sinuses with Ethmoid and Maxillary Infection, and Bronchiectasis: Kartagener's Triad, Tubercle 25:27-29 (March-April) 1944.

4. Siewert, A. K.: Über einen Fall von Bronchiectasis bei einem Patienten mit Situs Inversus Viscerum, Berl. klin. wchnschr. 41:139-141, 1904.

Choledochal Cyst Treated by Sphincterotomy

CARL E. ANDREWS, M.D., and WILLARD F. ANDE, M.D., West Palm Beach, Fla.

In recent years many case reports of choledochal cyst have appeared in the literature.[1-43] In general, it has been that the Roux en-Y drainage is the treatment of choice. Because of the rarity of this condition and the successful result of sphincterotomy, cholecystectomy, Cattell-tube drainage, and plastic closure of the common duct, it is felt wise to report this case.

Report of Case

A 44-year-old, married white woman was admitted to St. Mary's Hospital April 20, 1956, with a chief complaint of severe pain in the epigastrium. The patient had had recurrent bouts of right upper quadrant pain suggestive of gallbladder attacks, starting at the age of 10 years. There was no history of jaundice.

The patient had been known on previous roentgen examination to have nonvisualization of the gallbladder and "poor emptying of the stomach."

The pertinent findings on examination at the time of admission to St. Mary's Hospital were confined to the abdomen. There was marked muscle spasm in the epigastrium and right upper quadrant, with tenderness to pressure in this area. No definite mass could be felt. There was no costovertebral-angle tenderness. The liver, kidneys, and spleen were not palpable. A well-healed lower abdominal scar was noted, in keeping with the history of bilateral salpingo-oophorectomy and hysterectomy.

Laboratory work on admission revealed 4,840,000 red blood cells, 15,550 white cells per cubic millimeter, 14.5 gm. % (94%) of hemoglobin, 91% polymorphonuclear leukocytes, 4% lymphocytes, 47% stab forms and 44% segmented forms (Schilling count), 5% mononuclear cells. The urine was yellow and cloudy, with a specific gravity of 1.012. Albumin 1+, sugar negative, acetone negative, white cells 4-6 per cubic millimeter; epithelial cells few.

Serum amylase was 1,105 units (normal for the laboratory 80-200 units); cholesterol 288 mg. % (normal values for the laboratory 150-200 mg. %); total proteins 6.40 gm. %, albumin 4.45 gm. %, and globulin 1.95 gm. %; A/G ratio 2.2:1. This picture supported a diagnosis of acute cholecystitis and pancreatitis. On April 21 the total white cell count was 6,900, the young forms being reduced to 25 per cubic millimeters. Urinalysis on this day was negative for bile, bilirubin, and urobilinogen. On April 22 the serum amylase was 358 units.

A summary of the x-ray examinations by Dr. Hoover follows. "Chest examination negative; reexamination of the gallbladder failed to visualize, and the upper gastrointestinal tract presented only evidence of extrinsic pressure on the duodenal cap."

The patient's symptoms subsequently subsided. She refused to undergo exploration at this time. The discharge diagnosis was nonfunctioning gallbladder with chronic and subacute cholecystitis, probable cholelithiasis, and pancreatitis.

On April 27 the patient was readmitted to the hospital, after recurring bouts of pain. She was prepared for surgery, and at operation, through an upper abdominal right rectus incision, a few adhesions of the omentum to the lower abdominal midline scar were found. The gallbladder was of normal size and consistency, and there were no palpable stones. The pathology was in the common-duct area, where there was a large cystic mass medial and inferior to the gallbladder. The mass was about 2 in. (5 cm.) in diameter, and, as it was dissected out, a great many blood vessels were noted extending around it in a transverse fashion. The hepatic artery was anterior to the cystic mass at the proximal portion in the region of the hepatic ducts. The cystic mass was dissected approximately three-quarters of the way around, when it was elected to open this cyst. When the cyst was opened, clear bile was obtained. The cyst was further opened and explored. An operative cholangiogram was obtained after closing the opening around the catheter (Fig. 1). The hepatic-duct system was anomalous in its distribution, size, and shape. We believed we were dealing with a congenital cyst of the common duct. Because of the pancreatitis, a sphincterotomy was definitely indicated. The duodenum was mobilized, and it was opened longitudinally over a probe in the common duct. The sphincter was incised, and a No. 9 Bakes dilator was passed down through this opening. A 00 silk suture was tied around the dilator and drawn back into the cyst. The funnel-shaped gallbladder was then removed, and two very small pieces of gravel were visualized. The patient's

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Palm Beach Clinic (Dr. Andrews); Assistant Surgeon (Dr. Ande).

Fig. 1.—Cholangiogram taken in operating room. Note the anomalous hepatic ducts and the funnel-shaped gallbladder. The tube for injecting the dye enters the collapsed cyst. There is some leakage of dye, as seen about the distal common duct.

pain had undoubtedly been due to the passage of small gravel particles into the duodenum. Multiple small arteries extended into the gallbladder in addition to the main cystic artery. The latter was tied with 00 silk. The gallbladder emptied into the proximal portion of the cystic mass. There was a valve-like structure at the junction of the gallbladder and the cystic mass, but no definite cystic duct was encountered. This valve-like structure was opened and trimmed down flush with the cystic mass. The 00 silk was tied to a No. 16 Cattell tube, in which multiple openings had been made, and the Cattell tube was then pulled down into the duodenum through the duodenostomy. The excess portion of the cystic structure of the common duct was excised. A plastic closure of the common duct was performed with running 0000 chromic surgical gut over the Cattell tube, forming a long, funnel-shaped common duct. The duodenum was closed longitudinally. The gallbladder bed was closed, and drains were placed in the foramen of Winslow and brought out through a stab wound. The Cattell tube was brought out through a separate stab wound, and the abdomen was closed in layers. The patient received 1,500 cc. of blood during the operation, and she went to her room in good condition.

In the postoperative period there was considerable drainage for one week. The patient was discharged on May 10, 1956, on the 15th postoperative day.

Pathologic Report

(Dr. Hugh Dortch Jr., and Dr. V. M. Johnson)

"Clinical Diagnosis

"Preoperative: Acute cholecystitis. Postoperative: Congenital cyst of common duct.

"The specimen consists of gallbladder and tissue from inside a congenital cyst of the common duct.

"Gross Description

"The smaller specimen consists of an irregular mass of soft, friable, grayish-tan material, slightly wrinkled along its surface, which might be membranous exudate or might be very soft, friable tissue. It measured 2 cm. in maximum length.

"The second bottle contains a long, narrow gallbladder accompanied by three pieces of rather deeply congested membranous tissue, measuring up to 3 mm. in thickness and 5 cm. in diameter, their origin being uncertain. They might represent the wall of a cyst.

"The gallbladder is unusually slender, measuring 11×2.5 cm., and the diameter is only 1 to 2 cm. throughout most of its extent. It presents an edematous and deeply congested serosa, and the lumen contains a mixture of blood clot and mucoid material. The muscular wall is penetrated at many points by the mucosa, especially at the tip, so as to resemble the so-called adenomyoma. The rugae are markedly increased in thickness throughout the entire organ.

"Microscopic Description

"The wall of the 'cyst' consists of a mucosal surface somewhat resembling that of the gallbladder, except that it does not present prominent folds, surrounded by a rather thin smooth-muscle wall, in which three distinct layers are sometimes evident, and in which considerable scarring and some distortion have occurred. The muscular wall is much thinner than that of a gallbladder, and in structure it resembles a bile duct. The mucosa is ulcerated at many points, elsewhere consisting of a columnar epithelial layer, frequently arranged in a glandular pattern, with occasional glands of the Luschka type appearing in the deeper portions, near the muscular wall. A widely scattered infiltrate of lymphocytes is found at many points in the wall of the structure, often associated with the scar tissue, which has developed at all levels, sometimes becoming thickest outside the muscular wall; and a much more prominent acute inflammatory exudate is found involving all of the layers rather diffusely. This consists of scattered polymorphonuclear leukocytes appearing throughout the tissue spaces and becoming most concentrated on the internal and external surfaces of the specimen, where a fibrinopurulent exudate is often attached, appearing looser on the external surface. Numerous small

Fig. 2.—Cholangiogram on the fifth postoperative day.

nerves and vessels are found in the outer fibrous layer, and a small amount of fat is occasionally attached. Thus, the appearance is that of a scarred wall of a bile duct, and the size of the particles received and the surgical observation of the size and the shape of the lesion indicate that this probably does represent congenital cystic dilatation of the bile duct, or the so-called "choledochus cyst." The separate soft material proves to be a fibrinopurulent exudate, and this was apparently stripped from the external surface of the dilated duct.

"In the gallbladder we find moderate round-cell infiltration of the muscular wall, while the mucosa presents less extensive inflammatory infiltration, with considerable thickening of the mucosa, and formation of accessory glandular structures at various levels. This appearance is typical of chronic glandular proliferative cholecystitis. The thickened mucosa is composed of rather tall columnar epithelial cells, possessing abundant pale-staining lavender cytoplasm, and some of the small rounded glandular structures which have developed present some mucoid secretion. At many points Rokitansky-Aschoff sinuses have developed, projecting into and through the muscular wall, and at the apex of the organ larger glandular structures are found external to the muscular layer, but partially surrounded by thin muscle bundles. The over-all topography of this area is indicative of the hamartoma or malformation of the gallbladder known as an 'adenomyoma.'

"Diagnosis

"Congenital cystic dilatation of the common bile duct (choledochus cyst) with acute and chronic inflammatory reaction and scarring; chronic glandular proliferative cholecystitis; adenomyoma of the gallbladder."

Course

On May 19, 1956, a cholangiogram was done, showing an excellent biliary tract. (Fig. 2). The Cattell tube was removed on May 25 (one month after surgery). This was followed by some drainage at the Cattell-tube site for a week. However, the patient continued to feel fine and increased in strength, so that she was able to take a tour of Europe in June, 1956. An intravenous cholangiogram was accomplished 15 months after surgery (Fig. 3). Her general health is now excellent, 33 months after surgery. She has only mild gastrointestinal distress after eating nuts.

Fig. 3 (Dr. R. D. D. Hoover).—Intravenous cholangiogram on the 15th postoperative month.

A preliminary film is obtained over the gallbladder region, demonstrating no evident gross renal pathology, or evident opaque calculus of the gallbladder or renal region.

Intravenous cholangiography is performed, and on the subsequent film study, at 10 minutes, there is appearance of the dye both in the duct system of the liver and in the right renal structure, with increasing concentration thereafter in the biliary duct system and decreasing renal concentration. The combined study demonstrates that there is excellent excretion of the dye, without obstruction to the distal common duct, as the dye is noted to pass freely into the duodenum. Insofar as can be determined, there is no evident obstruction, calculus, or specific disease process.

Conclusion: Normal visualization and function.

Summary

A case of choledochal cyst, anomalous hepatic ducts, pancreatitis, and adenomyoma of the gallbladder is reported. The treatment consisted of sphincterotomy, cholecystectomy, Cattell-tube drainage, and plastic closure of the common duct. Each cyst should be evaluated by cholangiography and direct inspection. If there are (1) adequate blood supply, (2) absence of gastric mucosa,[25] and (3) a good-sized common duct distal to the cyst,[23] then sphincterotomy, cholecystectomy, and plastic closure with a Cattell tube will give an excellent result.

Palm Beach Clinic, 705 N. Olive Ave.

REFERENCES

1. Alden, J. F., and Sterner, E. R.: Diverticulum of the Common Bile Duct, Ann. Surg. 145:269, 1957.

2. Archambault, H.; Archambault, R., and Lasker, G. W.: Choledochus Cyst: A Case Treated by Y-Roux Type Anastomosis of Jejunum to the Cyst, Ann. Surg. 132:1144, 1950.

3. Attar, S., and Obeid, S.: Congenital Cyst of the Common Bile Duct: A Review of the Literature and a Report of 2 Cases, Ann. Surg. 142:289, 1955.

4. Austin, H. F.: The Blood Supply of the Common Bile Duct and Its Relationship to Duodenum, Brit. J. Surg. 43:75, 1955.

5. Barbosa, E., and Rate, R. G.: Cystic Dilatation of the Common Duct: Review of the Literature and Report of a Case, J. Internat. Coll. Surgeons 25:430, 1956.

6. Bernheim, M.; Marion, P.; Roux, J.-A.; Lanternier, J., and Bethenod, M.: Congenital Cysts of the Choledocus in the Nursling, Pédiatrie 8: 677, 1953.

7. Blegen, H. M., and Boyer, E. L.: Perforation of Choledochus Cyst with Biliary Peritonitis, Journal-Lancet 66:177, 1946.

8. Blocker, T. G., Jr.; Williams, H., and Williams, J. E.: Traumatic Rupture of the Congenital Cyst of the Choledochus, Arch. Surg. 34:695, 1937.

9. Bowen, F. H.: Choledochal Cyst, J. Florida M. A. 44:1099, 1958.

10. Brooks, B., and Weinstein, A.: Cyst of the Ampulla of Vater, Ann. Surg. 117:728, 1943.

11. Cantey, W. C.: Congenital Cystic Dilatation of the Common Bile Duct, Ann. Surg. 143:608, 1956.

12. Dennison, W. M.: Choledochus Cyst in Childhood, J. Internat. Coll. Surgeons 21:113, 1954.

13. Dickinson, E. H., and Spencer, F. C.: Choledochal Cyst: Report of a Case with Unusual Features, J. Pediat. 41:462, 1952.

14. Goldman, W. P., and Zack, J. J.: Choledochus Cyst, Canad. M. A. J. 68:162, 1953.

15. Gordimer, H., and Bluestone, L.: Enterogenous Cyst of the Duodenum: Case Report, Ann. Surg. 132:1149, 1950.

16. Gross, R. E.: The Surgery of Infancy and Childhood, Philadelphia, W. B. Saunders Company, 1953.

17. Grove, W. J.: Recognition and Treatment of Congenital Choledochal Cysts, A. M. A. Arch. Surg. 75:443, 1957.

18. Gupta, S. C.: Congenital Anomalies of the Biliary Apparatus, Indian J. Surg. 11:231, 1949.

19. Hamilton, I.: Congenital Cystic Dilatation of the Common Bile Duct, M. J. Australia 1:800, 1939.

20. Hertzler, J. H., and Maguire, C. E.: Congenital Dilatation of the Common Bile Duct: Report of 2 Cases in Children, A. M. A. Arch. Surg. 62:275, 1951.

21. Irwin, S. T., and Morison, J. E.: Congenital Cyst of the Common Bile-Duct Containing Stones and Undergoing Cancerous Change, Brit. J. Surg. 32:319, 1944.

22. Keeley, J. L.: Congenital Cystic Dilatation of the Common Bile Duct, Arch. Surg. 56:508, 1948.

23. Keith, L. M., Jr.; Rini, M., and Martin, L. H.: Congenital Cystic Dilatation of the Common Duct (Choledochal Cyst), A. M. A. Arch. Surg. 75:143, 1957.

24. Kjellberg, S. R.: Water Soluble Contrast Medium for Precise Roentgenological Diagnosis of Cysts of the Common Bile Duct, Acta radiol. 29:307, 1948.

25. Lee, G. M., Jr.: Duplication of the Cystic and Common Hepatic Ducts, Lined with Gastric Mucosa, New England J. Med. 256:927, 1957.

26. Lumpkin, F. E., Jr., and Wilson, J. W.: A Method of Diagnosis of Choledochal Cyst, Am. Surgeon 21:63, 1955.

27. Rheinlander, H. F., and Bowens, O. L., Jr.: Congenital Absence of the Gall Bladder with Cystic Dilatation of the Common Bile Duct, New England J. Med. 256:557, 1957.

28. Serfas, L. S.; Lyter, C. S.: Choledochal Cyst, with a Report of an Intraduodenal Choledochal Cyst, Am. J. Surg. 93:979, 1957.

29. Shallow, T. A.; Eger, S. A., and Wagner, F. B., Jr.: Congenital Cystic Dilatation of Common Bile Duct: Case Report and Review of Literature, Ann. Surg. 117:355, 1943.

30. Shallow, T. A.; Eger, S. A., and Wagner, F. B., Jr.: Congenital Cystic Dilatation of Common Bile Duct, Follow-Up on Previously Reported Case and Report of Additional Case, Ann. Surg. 123:119, 1946.

31. Shocket, E.; Hallenbeck, G. A., and Hayles, A. B.: Choledochal Cyst: Report of Cases, Proc. Staff Meet. Mayo Clin. 30:83, 1955.

32. Strohl, E. L., and Sarver, F. E.: Cystic Dilatation of the Common Bile Duct. A. M. A. Arch. Surg. 62:597, 1951.

33. Swartley, W. B., and Weeder, S. D.: Choledochus Cyst with a Double Common Bile Duct. Ann. Surg. 101:912, 1935.

34. Tsardakas, E., and Robnett, A. H.: Congenital Cystic Dilatation of the Common Bile Duct: Report of 3 Cases, Analysis of 57 Cases, and Review of the Literature, A. M. A. Arch. Surg. 72: 311, 1956.

35. Walters, W.: Physiologic Studies in Cases of Stricture of the Common Bile Duct, Ann. Surg. 130:448, 1945.

36. Weeder, S. D.: Choledochus Cyst with a Double Common Bile Duct: Sequelae and Complications, Ann. Surg. 110:55, 1939.

37. Wheeler, W. I., de C.: Unusual Case of Obstruction to Common Bile-Duct (Choledochocele?), Brit. J. Surg. 27:446, 1940.

38. Williams, C., and Williams, A. M.: Abnormalities of the Bile Ducts, Ann. Surg. 141:598, 1955.

39. Wrightson, P.: Congenital Cystic Dilatation of the Common Bile Duct: A Case Occurring in Infancy, Australian & New Zealand J. Surg. 23: 110, 1953.

40. Healey, J. E., Jr., and Schroy, P. C.: Anatomy of the Biliary Ducts Within the Human Liver: Analysis of the Prevailing Pattern of Branchings and the Major Variations of the Biliary Ducts, A. M. A. Arch. Surg. 66:599, 1953.

41. Alonso-Lej, F.; Rever, W. B., Jr., and Pessagno, D. J.: Congenital Choledochal Cyst, with a Report of 2, and an Analysis of 94, Cases, Collective Review, Internat. Abstr. Surg. 108:1, 1959, in Surg. Gynec. & Obst., Jan., 1959.

42. Ravitch, M. M., and Snyder, G. B.: Congenital Cystic Dilatation of the Common Bile Duct: Special Reference to Radiographic Studies, Pediat. Surg. October, 1958.

43. Warren, K. W., and Polk, R. C.: Benign Cysts of the Liver and Biliary Tract, S. Clin. North America, p. 707, June, 1958.

Papillary Adenoma of the Ampulla of Vater

JAMES A. ROCK, M.D., and EDWIN R. FISHER, M.D., Pittsburgh

Benign neoplasms of the ampulla of Vater are rare. Chu,[1] in 1950, reported a case of an adenoma in the ampulla of Vater and reviewed the world literature of benign tumors within the extrahepatic biliary tree. He was able to collect only 29 documented examples up to that time. Since then, only a few additional reports have been added.[2-9] While the majority of these tumors have been epithelial in origin, variously designated as papillomas, polyps, adenomas, and carcinoid tumors, such mesenchymal neoplasms as fibromas, lipomas, neuromas, leiomyomas, and granular-cell myoblastomas have been noted in this region.

Although almost all of these tumors involved the common duct or adjacent areas of the extrahepatic biliary tree, only eight of the neoplasms were adenomas or papillomas of the ampulla of Vater. These produced sufficient symptoms to warrant surgical intervention. In no instance was the correct diagnosis made prior to operation.

Report of a Case

The patient, a 70-year-old white man, was admitted to the Veterans Administration Hospital, Pittsburgh, with a history of anorexia, malaise, nausea, and vomiting of three months' duration and a 40 lb. weight loss. There was no hematemesis.

Physical examination revealed icterus of the skin and sclerae. Other significant findings were confined to the abdomen, which was soft, flat, and nontender. The smooth edge of the right lobe of the liver was palpable 5.0 cm. below the right costal margin. A nontender mass, measuring approximately 4 cm. in diameter, was felt in the left upper quadrant. Hemograms were persistently within normal limits, while the alkaline phosphatase was 12.0 Bodansky units and the serum bilirubin 6.94 mg. %. Cephalin flocculation was 2+ at 24 hours and 3+ at 48 hours, and the sulfobromophthalein (Bromsulphalein) test revealed retention of 47% of the dye at 45 minutes. There was little variation in the prothrombin time, and the serum amylase was 32 glucose equivalents. X-ray examination of the gastrointestinal tract was normal, while a iodipamide (Cholografin) study failed to visualize the hepatic system. Also, a splenoportogram failed to outline the splenic or portal vessels. The urine urobilinogen was 3.6 E. U. and the fecal urobilinogen was 41.0 E. U. per 100 mg.

An exploratory laparotomy was performed, and a firm mass in the ampullary region was palpated. During the operation the patient experienced a marked drop in blood pressure, and a cholecystojejunostomy was performed with the intent of subsequently performing a more radical procedure.

On the sixth postoperative day the serum bilirubin was 1.27 mg. % (direct 0.75 mg. % and indirect 0.52 mg. %), and by the 22d postoperative day the alkaline phosphatase was 7.94 Bodansky units and the sulfobromophthalein retention only 6.0%. The patient showed a gradual weight gain and general clinical improvement. Another laparotomy was done one month later, and duodenectomy with subtotal pancreatectomy and excision of the distal portion of the common duct was performed. In addition, subtotal gastrectomy, gastrojejunostomy, choledochojejunostomy, and pancreaticojejunostomy were done. Except for a transient elevation of the serum amylase to 570 glucose equivalents, the postoperative course was uneventful.

Pathologic Findings

Gross Examination.—The surgical specimen consisted of the distal portion of the stomach, which was contiguous with a 23.0 cm. length of duodenum, together with the head of the pancreas and the distal portion of the common bile duct. In addition, a segment of distal duodenum 10.0 cm. in length was also submitted. The ampulla of Vater was 0.5 cm. long and completely occluded by numerous folds of soft, glistening, dark purplish-red tissue, which extended for a distance of 1.0 cm. up the common duct and protruded slightly into the duodenum, where the adjacent intestinal mucosa was redundant. The common

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From the Departments of Pathology, University of Pittsburgh School of Medicine and Veterans Administration Hospital.

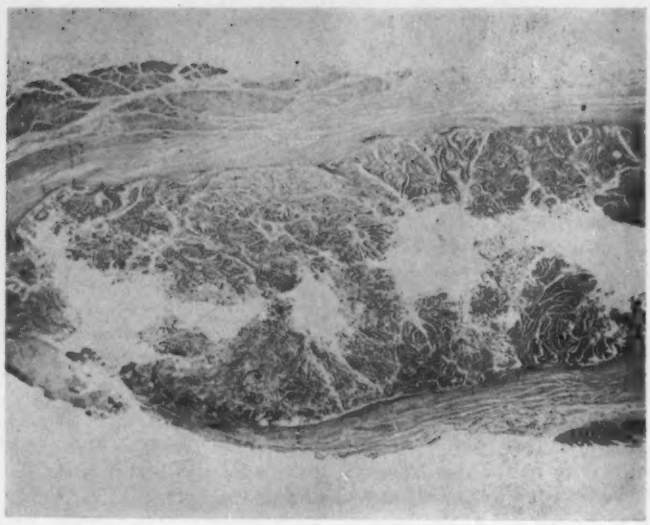

Fig. 1.—Low-power appearance of neoplasm, revealing intraluminal growth only. ×8.

duct was moderately dilated, and the wall of the pancreatic duct was thick. The separate segment of duodenum was unremarkable.

Microscopic Examination. — Multiple sections through the ampulla of Vater revealed a benign epithelial neoplasm formed by columnar epithelial cells arranged on fibrous and vascular stalks in a papillary fashion (Fig. 1). The stroma was diffusely infiltrated with plasma cells, lymphocytes, and eosinophils. The tumor cells were uniform and without loss of polarity (Fig. 2*A* and *B*). Their nuclei were elongated, and their abundant cytoplasm was eosinophilic and finely granular. Mitoses were rare, and multiple sections failed to show invasion into the muscularis of the ampulla. Sections of the other tissues were not remarkable except for slight dilatation and thickening of the pancreatic ducts.

Comment

Baggenstoss,[10] in his review of the ampullar region in 100 consecutive autopsies, found 4 polyps in the ampulla of Vater, while a review of all autopsies performed at the Mayo Clinic up to 1936 revealed only 29 polyps in this region. This indicates that these lesions may be commoner than is apparent from previous reports and suggests that an accurate estimate of their incidence may depend upon more careful inspection of this area at necropsy. Such an estimate may be especially important in view of the report by Cattell and Pyrtek,[11] who reported four instances of carcinoma of the ampulla of Vater which they considered arose from benign adenomatous lesions. In some of these cases the superficial areas of the neoplasms demonstrated a benign histologic pattern, while deeper sections showed considerable dysplasia and invasion of the submucosa. Such an event is more commonplace in adenomatous lesions observed in other portions of the gastorintestinal tract, particularly the colon and stomach. Indeed, Fisher and Castro[12] found foci of malignancy in each of four diffuse papillary adenomas of the colon after study of step sections of serial blocks. In the case presented here, numerous sections were made throughout the entire neoplasm and adjacent structures without finding cytologic evidence of malignant transformation or invasion into the muscularis. The prognosis in this case should be excellent. Nevertheless, if analogies between these adenomatous lesions and those of the colon and stomach are valid, one might suspect that papillary adenomas of the ampulla of Vater are premalignant lesions. Evaluation of these lesions by means of only a few superficial, random biopsies may not reveal their true nature and may lead to inadequate therapy.

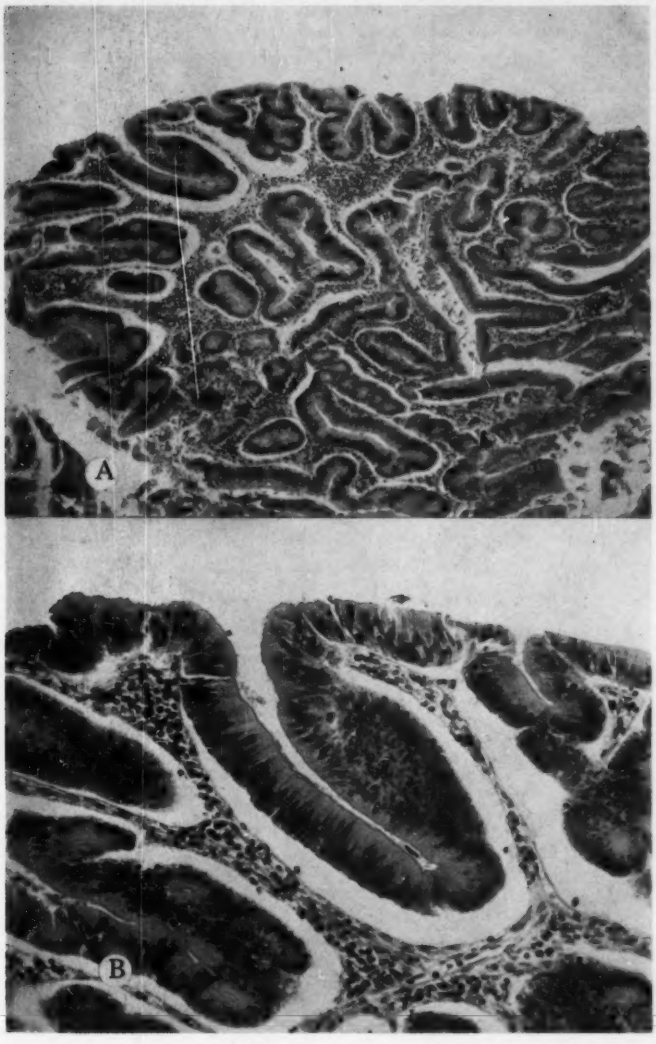

Fig. 2.—Adenomatous configuration of neoplasm is apparent and, although reduplication of lumina is prominent (*A*; ×85), absolute criteria of malignancy are lacking (*B*; ×235).

Summary

An example of papillary adenoma of the ampulla of Vater is presented, and the rarity of this lesion is indicated. The relationship of this lesion to carcinoma at this site is discussed.

Veterans Administration Hospital, University Drive (Dr. Rock).

REFERENCES

1. Chu, P. T.: Benign Neoplasms of the Extrahepatic Biliary Ducts, Arch. Path. 50:84-97, 1950.

2. Archambault, H., and Archambault, R.: Leiomyoma of the Common Bile Duct, A. M. A. Arch. Surg. 64:531-534, 1952.

3. Smith, H. P., Jr., and Blakemore, W. S.: Benign Polyp of the Ampulla of Vater, Radiology 56:571-574, 1951.

4. Moore, S. W.; McElwee, R. S., and Romiti, C.: Benign Tumors of the Biliary Tract, J. A. M. A. 150:999-1002, 1952.

5. Coggins, R. P.: Granular-Cell Myoblastoma of Common Bile Duct, A. M. A. Arch. Path. 54: 398-402, 1952.

6. Duncan, J. T., Jr., and Wilson, H.: Benign Tumor of the Common Bile Duct, Ann. Surg. 145:271-274, 1957.

7. Keech, M. K.: Cystadenomata of the Pancreas and Intrahepatic Bile Ducts, Gastroenterology 19:568-574, 1951.

8. Rognon, L. M.: Adenoma of Ampulla of Vater, Mém. Acad. chir. 81:504-509, 1955.

9. van der Zwaag, G. L.: Benign Tumours of the Bile Ducts, Arch. chir. neerl. 8:102-106, 1956.

10. Baggenstoss, A. H.: Major Duodenal Papilla: Variations of Pathologic Interest and Lesions of the Mucosa, Arch. Path. 26:853-868, 1938.

11. Cattell, R. B., and Pyrtek, L. J.: Premalignant Lesions of the Ampulla of Vater, Surg. Gynec. & Obst. 90:21-30, 1950.

12. Fisher, E. R., and Castro, A. F.: Diffuse Papillomatous Polyps (Villous Tumors) of the Colon and Rectum: Pathologic and Clinical Observations, Am. J. Surg. 85:146-151, 1953.

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1. Perkins, J. L.: Kansas State M. J. (to be published).

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1. Rand, N. T. and Collins, V. K.: Food Technology *12*:585 (Nov.) 1958.

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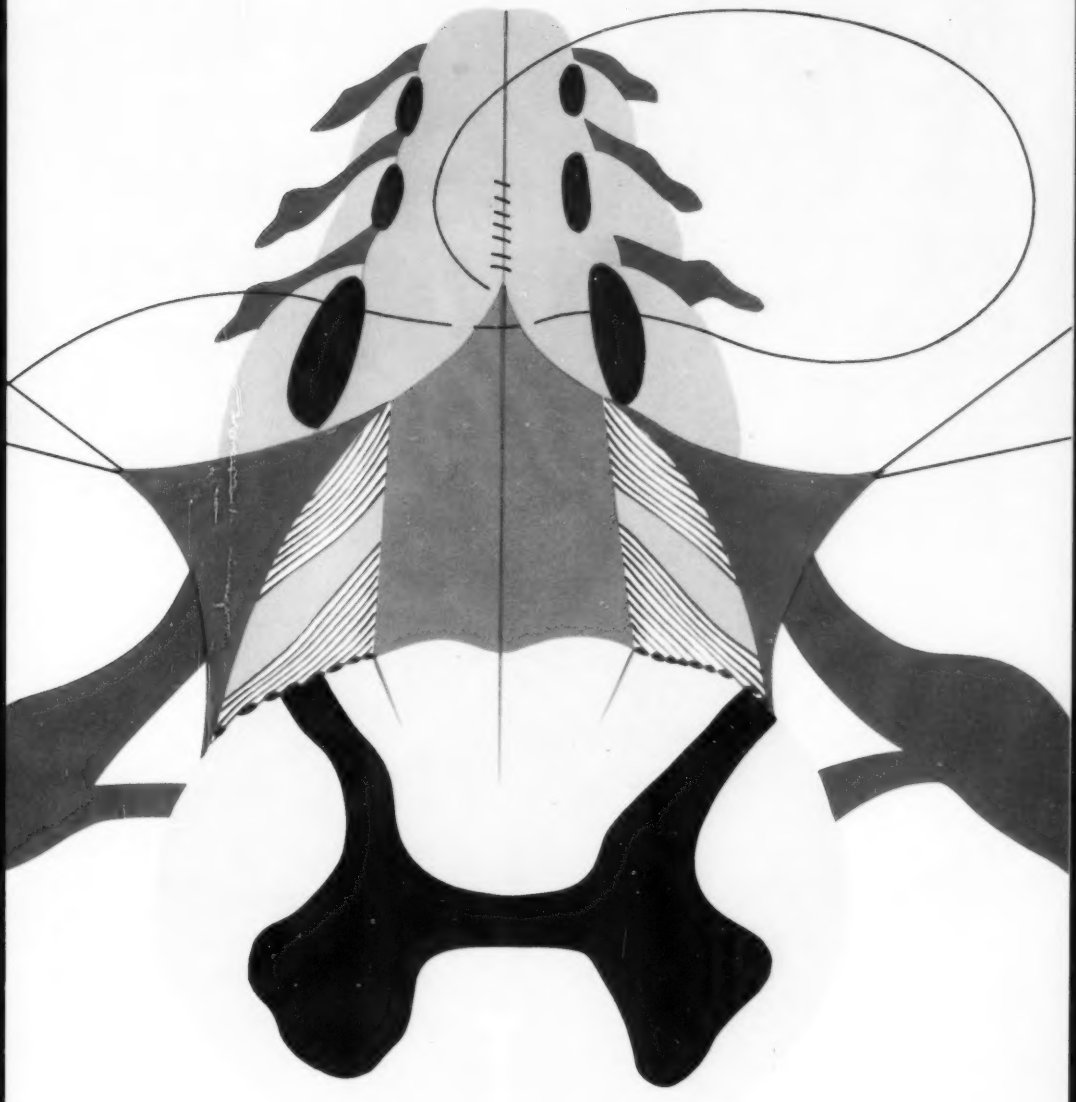

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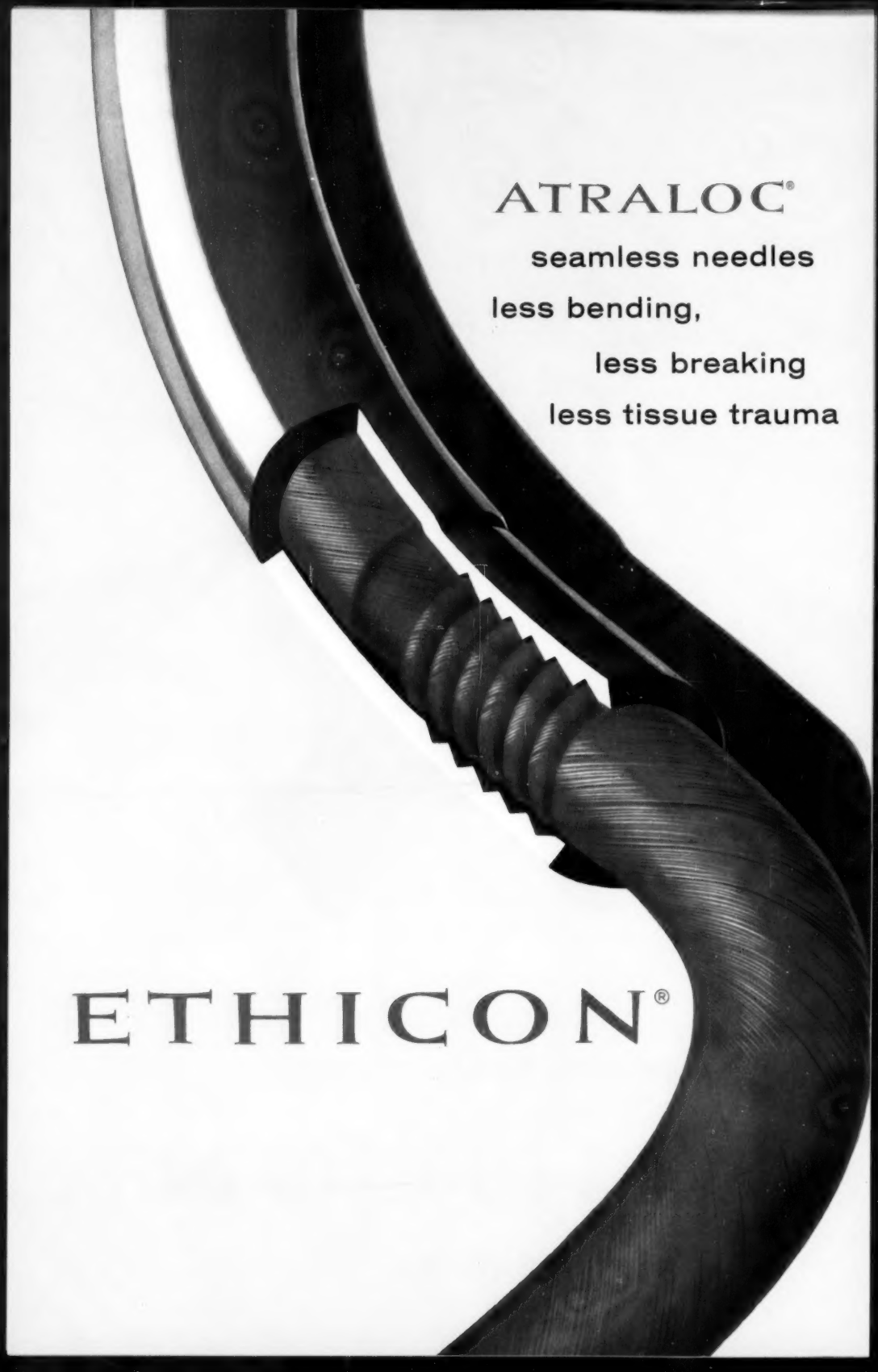
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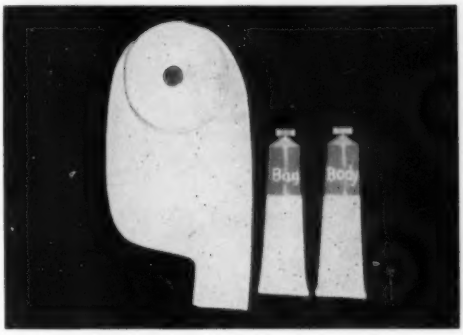

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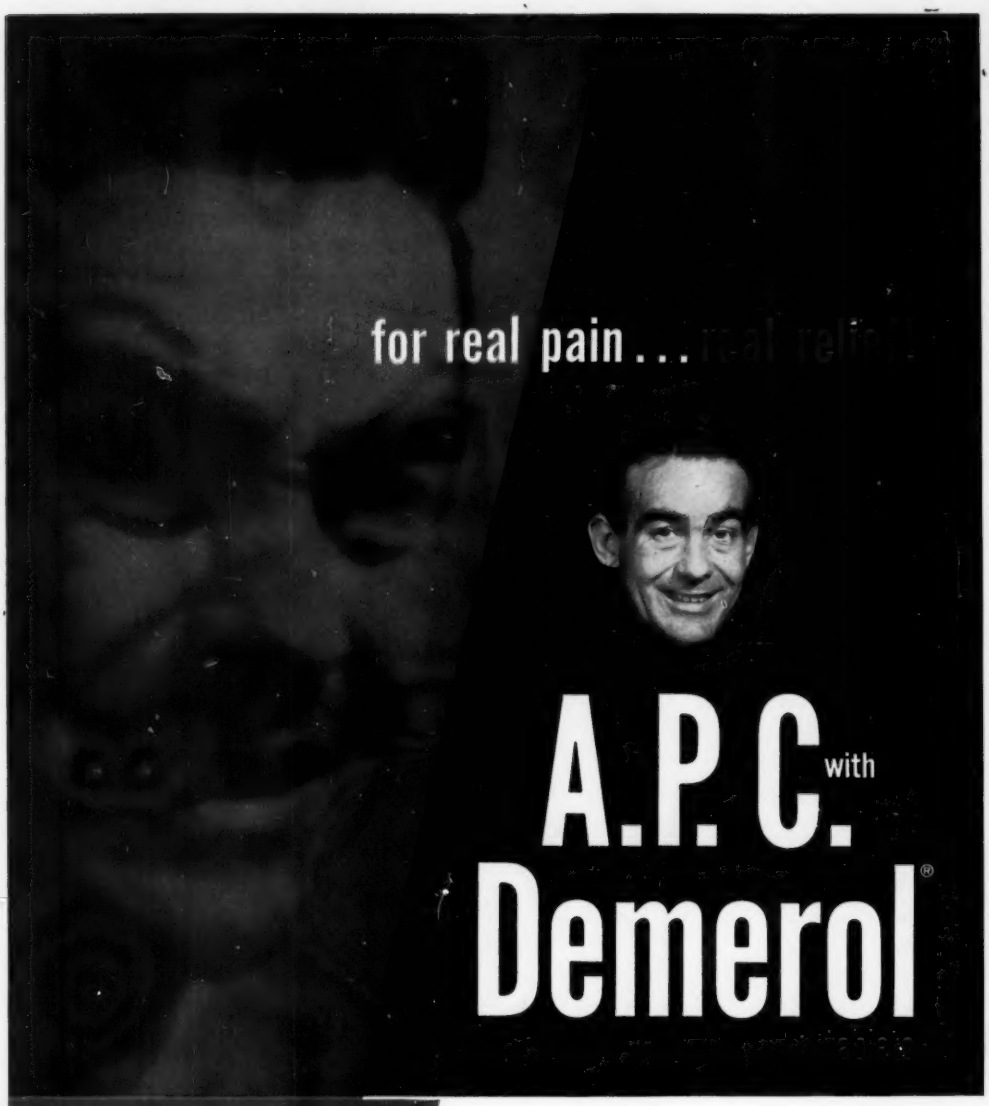

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